THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

OCTOBER, 1933

COMMUNICATIONS

THE OCULAR CONDITIONS IN DIABETES MELLITUS

BY

W. A. GRAY

LONDON

IN this investigation the incidence of retinitis, vitreous haemorrhage, and ocular palsies was determined in five hundred cases of diabetes mellitus. In 228 of these patients the number showing arterio-sclerosis of the retinal vessels and cataract, visible with an ophthalmoscope, was assessed. The state and reactions of the pupil were examined in seven cases of coma, in twenty-four cases of hypoglycaemia (after overdose of insulin), and in twenty cases of diabetes mellitus with marked acidosis. An additional eighty patients were examined, under full mydriasis, with the Gullstrand slit-lamp to decide whether the incidence of cataract and early lens changes was more common in diabetics than healthy persons. The total number of diabetic cases examined was 583. True diabetic cataract occurred in only two instances out of the whole number of patients. In the seven cases of diabetic coma the intra-ocular tension was recorded.

Arterio-sclerosis of the Retinal Vessels.—The insulin epoch in diabetes with its successful treatment of coma has brought into prominence the importance of arterio-sclerosis in the prognosis of diabetes mellitus. The diagnosis of vessel change is in many cases doubtful; and a knowledge of the frequency of association of retinal arterio-sclerosis with similar changes in other parts, would appear to be of value.

The arterio-sclerotic changes in the retinal vessels have been fully described by Gunn (1898) and Foster Moore (1916). Gunn's original signs were as follows:—(1) Irregularity of the lumen of the retinal arteries; (2) tortuosity of the arteries; (3) an exceptionally narrow and bright central light streak which may show a series of brighter points at intervals. These changes are of chief importance when they involve the tertiary and secondary branches of the retinal artery. (4) Loss of translucency of the arterial walls; (5) obstruction of the blood flow in the veins where they are crossed by arteries; (6) oedema of the retina. Foster Moore confirmed these observations with the exception of (6). He also pointed out that when a vein crosses under a sclerotic artery its course is deflected.

In diabetes the changes in the retinal vessels are seldom so pronounced as in simple arterio-sclerosis. The earliest sign observed was a loss of translucency of the arterial wall, so that at points of crossing the vein lying beneath the artery could not be seen as is usually possible in normal subjects. This was the only change in five cases and in them the diabetes had lasted ten months. A more frequent change was a slight deflection of the line of a vein where it was crossed by an artery. Normally the vein continues its course without deviation while passing under the artery. The angle of deflection was never pronounced as occurs in pure arterio-sclerosis. Alteration in the breadth of the vein on the distal side of the artery, tapering while beneath the artery to regain its normal size beyond, was seen. At the crossing the fine white lines described by Gunn and due to an increase in fibrous tissue of the vessel walls were evident in many cases but this appearance was limited and never extended for any distance along the course of the vessels. Irregularity in the lumen of the arteries causing an appearance in the macular region of a corkscrew-like effect in the vessels was never seen. The vessel changes in diabetes on the whole, differ from other arterio-sclerotic retinae in being less marked.

Clinical aspects of the cases with Sclerotic Retinal Vessels.—Age.—Of the 228 patients, 49 were below 35 years, and showed no change in the retina, no matter how long the diabetes had lasted (in some cases over six years). The remainder, 179 in number, were divided into the following age groups and the occurrence of the sclerosis noted.

Age		Number with retinal sclerosis
1. Between 35-45	...	13 with retinal sclerosis out of 31
2. Between 45-55	...	31 with retinal sclerosis out of 71
3. Between 55-65	...	34 with retinal sclerosis out of 57
4. Above 60	...	14 with retinal sclerosis out of 20

This table shows an increasing incidence of sclerosis of the retina with age, which suggests that diabetes expedites degenerative changes in the vascular system. It is not, in itself, the sole factor, for there is an absence of any change in the young. The average duration[1] of the diabetes in this group was two years and eight months. In those with normal retinae, the diabetes had lasted, on an average, for four years, and the majority were young patients, showing that there is probably some factor other than diabetes responsible for starting the vascular changes.

Severity of the Diabetes.—It is generally agreed that severe diabetes is usually unaccompanied by any retinal change (excepting the extremely rare lipaemia retinalis), for it occurs mainly in the young. The presence of diacetic acid and acetone was three times more common in those with normal retinae. These bodies have apparently no effect on the retina. Sixty-three of the cases with changes in the retinal vessels had a blood sugar[2] of above 2 per cent., while the normal is .08 per cent. to 0.12 per cent. This shows that well-marked cases of diabetes have frequently retinal sclerosis. They had been under treatment for two-and-a-half years with gradual increase in insulin.

Sclerotic vascular changes may be limited to one area of the body. Thus Clifford Allbutt pointed out that the arteries of the limbs may be much thickened, while those of the brain may be normal. Joslin holds this to be true of some cases of diabetes. Thus in young diabetics thickening of the arteries in the lower limbs has frequently been recorded while the retinal vessels showed no change. These observations were made before the days of insulin, for now autopsies on children are a rarity. In the present series of ninety-two patients with retinal vessel change, fifty-five had peripheral arterio-sclerosis, the percentage in the older patients being the highest. Spalding and Curtis had forty-six cases of arterio-sclerosis of the retina and in thirty-six of them similar changes occurred in the peripheral vessels. In Grafe's experience, based on von Noorden's patients, "every diabetic of eight years standing with high blood pressure invariably shows retinitis" and consequently arterio-sclerosis.

The difficulty of making a precise diagnosis in the case of changes in the peripheral vessels forced one to assess the frequency of the association with retinal sclerosis by taking more definite standards of vessel involvement elsewhere, *e.g.*, incidence of gangrene and heart affections. Gangrene occurred in sixteen of our cases. Amputated limbs of patients with diabetic gangrene always show extensive vascular involvement, and it is generally

[1] Approximate, for in old patients diabetes can exist for some time without symptoms.

[2] Fasting.

admitted that this is the essential cause of the condition. The average age of our patients with gangrene was fifty-two. Four had normal retinal vessels, retinitis occurred in five cases, retinal sclerosis in three others, while in the remaining four the fundus oculi could not be seen on account of co-existing cataract. The patients with retinitis and gangrene, showed only slight changes in the retinal vessels, such as loss of translucency of the arterial wall and a slight amount of deflection of the line of a vein at places where it crossed beneath an artery. The amount of sclerosis of the retina bore no relation to the severity of the peripheral change. Thus in a patient with diabetes who had marked calcification of the vessels of the limbs, the retinal vessels bore little evidence of sclerosis (Fig. 4). Haemorrhages of the retina developed in the course of a year. It is interesting, in connection with this case, to note that Willis states that since haemorrhage can occur although the vessels look normal, it is questionable whether

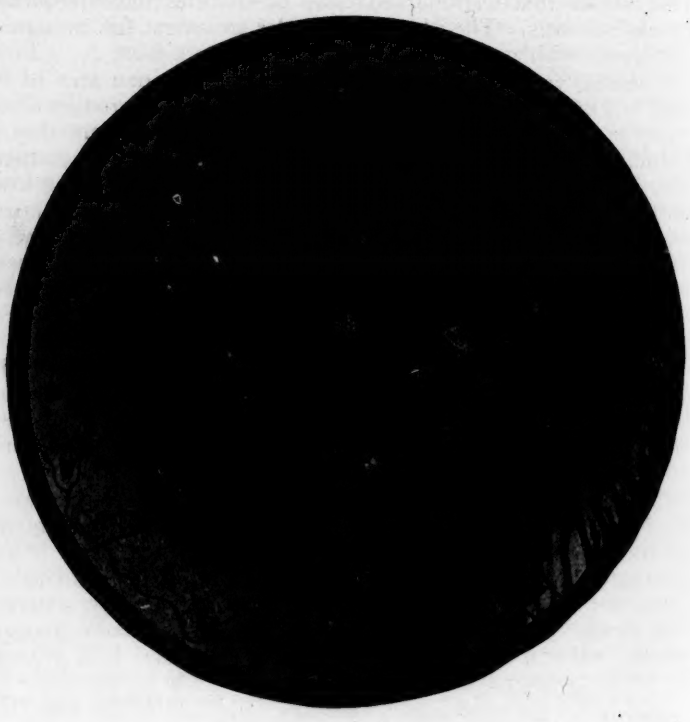
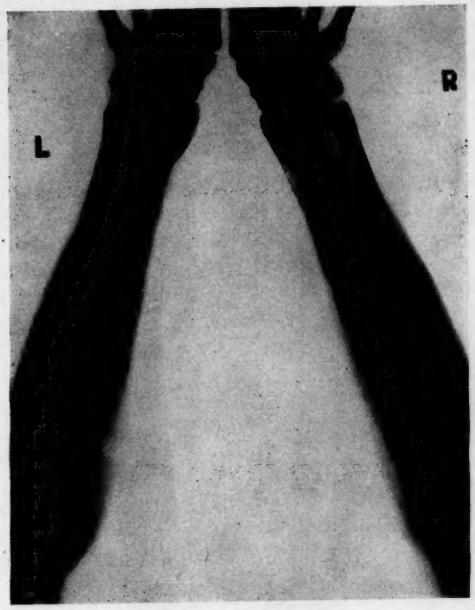
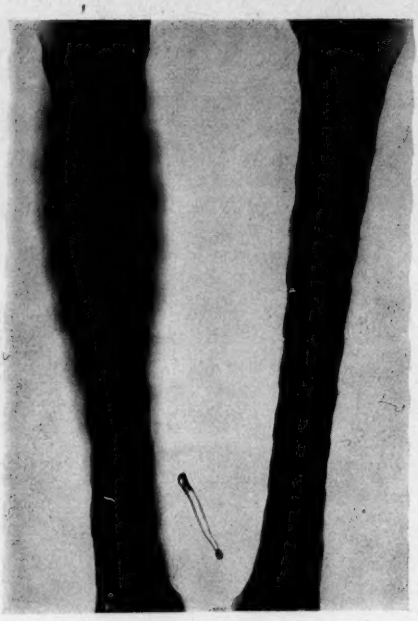


FIG. 4.


L
R



changes may not exist in the vessels without much external evidence. In three other cases of the present series with threatened gangrene of the toes, there was a greater degree of retinal sclerosis than in the patient with calcification of the peripheral arteries.

Heart Affections.—Coronary thrombosis was present in two cases, both men. One of these was 44 years of age, diabetic since 1923, on 40 units insulin per diem, blood sugar 0.15 per cent., only mild retinal sclerosis was found in this patient. The other patient was aged 68 years, with a mild form of diabetes of eighteen month's duration. He had, however, more marked retinal sclerosis than the first patient. The difference in age might be sufficient to account for the greater change in the retina, for, as has been shown, the older the patient, the greater the degree of sclerosis.

Cerebral Haemorrhage.—A definite relationship between arterio-sclerosis of the retinal vessels and those of the brain has been established by Foster Moore. In a disease such as diabetes which now so frequently starts in the arterio-sclerotic period of life, it might be expected that cerebral lesions would be fairly common. Examination of the causes of death in 673 patients at the London Hospital showed that out of 22 deaths, three were due to cerebral

haemorrhage, nine to coma, four to gangrene, four to heart failure, and the remainder to infection. These figures show the relative importance of arterio-sclerosis as a cause of death compared with coma.

Conclusions

The incidence of retinal sclerosis is higher the older the patient.

A definite relationship exists between arterio-sclerosis of the retina and the other parts of the body.

Even slight evidence of vessel change in the retina is important, since it is highly probable that more pronounced changes may exist elsewhere.

Section II—Retinitis

Historical.—Retinitis in diabetes mellitus has long been a recognized condition. Edward Jaeger published the first report in 1856. He was followed by Desmarres (1858), Noyes (1869), Haltenhoff (1873), Leber (1875), Mackenzie (1877), Nettleship (1877), who described cases of retinitis in diabetes mellitus unaccompanied by albuminuria. Nettleship in 1877 carefully described the histological changes in the retina. Parsons (1904) pointed out the difference between diabetic and other forms of retinitis and Foster Moore showed that it can occur in a distinctive form.

Occurrence.—In the present investigation sixty-six cases of retinitis were observed in five hundred diabetics. Other figures are those of von Noorden, 81 out of 477 cases of diabetes, and Spalding and Curtis, 16 in 307 cases.

Retinitis cases were divided into the following groups:—

1. Appearance:
   - (*a*) Exudates only.
   - (*b*) Haemorrhages only.
   - (*c*) Haemorrhages and exudates.
   - (*d*) Large retinal haemorrhages.
   - (*e*) Advanced type.
2. Condition of the retinal vessels.
   - (*a*) General changes.
   - (*b*) Pressure changes of retinal vessels.
     - (i) Hypotension.
     - (ii) Hypertension.

3. Significance of retinitis.
   (*a*) Differentiation from retinitis of other origin.
   (*b*) Severity of the diabetes.
   (*c*) Aetiology of the haemorrhages.

In addition to the 66 cases to be described, there were five other cases of retinitis occurring in diabetics, associated with albuminuria and arterio-sclerosis. As they did not present any characteristics which could be attributed to diabetes mellitus, they are omitted from the following description.

Appearances. (*a*) *Exudates only.* (Fig. 1).

There were five cases showing exudates only, of which Fig. 1 is an example. These exudates have the appearance of yellowish white spots and always appeared first near the macula. They

FIG. 1.

were unaccompanied by haemorrhages and are in their appearance, primary situation and their method of spread, the most characteristic lesion of diabetic retinitis. They spread in a regular manner outwards from the macula, forming an incomplete ring round it, and for a time are restricted to the area of the retina bounded by the upper and lower temporal vessels. This spread can be traced in Fig. 3. When they reach the temporal vessels, they abut against the main vessel walls. They increase in size with age by coalescing. These yellowish white spots occurred in three cases in one eye only and remained unilateral for an average period of one year. They are the earliest specific sign of a diabetic retinitis.

Accompanying Conditions.—The average age of the patients was 51 years, and the duration of diabetes was almost three years. Three of them were under insulin treatment. Sclerotic vessel changes were present and there was hypotension of the retinal arteries. None of the patients had albuminuria and septic lesions were absent. It may be questioned whether these cases of exudate alone are arterio-sclerotic. The sclerotic changes in the retinal vessels were slight. Also in three cases they remained unilateral for some time. Against this must be cited the absence of any pronounced arterio-sclerotic change in the fundus vessels, which is a feature of a pure arterio-sclerotic retinitis (Foster Moore) where retinal haemorrhages are almost always present. In addition in diabetes the spots are limited to the central area of the retina corresponding to retinitis punctata centralis of Hirschberg, who described the occurrence of exudates and haemorrhages in this area as the most typical type of diabetic retinitis.

(*b*) *Haemorrhages only.* (Fig. 2).

Haemorrhages alone were present in sixteen patients, of which Fig. 2 is an example. The haemorrhages were round or roughly triangular, deeply situated, occurring first at the periphery of the fundus, outside the main temporal vessels, and gradually spreading into the macular area, which was last affected. A characteristic point was that the haemorrhages were situated at a regular distance, about 3 mms., away from the main vessel trunks, and never encroached beyond this distance. When the whole fundus is affected the yellowish-white exudates begin to appear at the macula (Fig. 5). Once a haemorrhage occurred, it persisted but did not increase in size. This type thus contrasts with the first type (*a*) in the site of their first appearance and manner of spread and relation to main vessels. They were always bilateral. The associated conditions were—average age was 56 years, duration of diabetes was three and a half years. Two were under insulin and the remainder on diet only. Six of them had albumen and three had

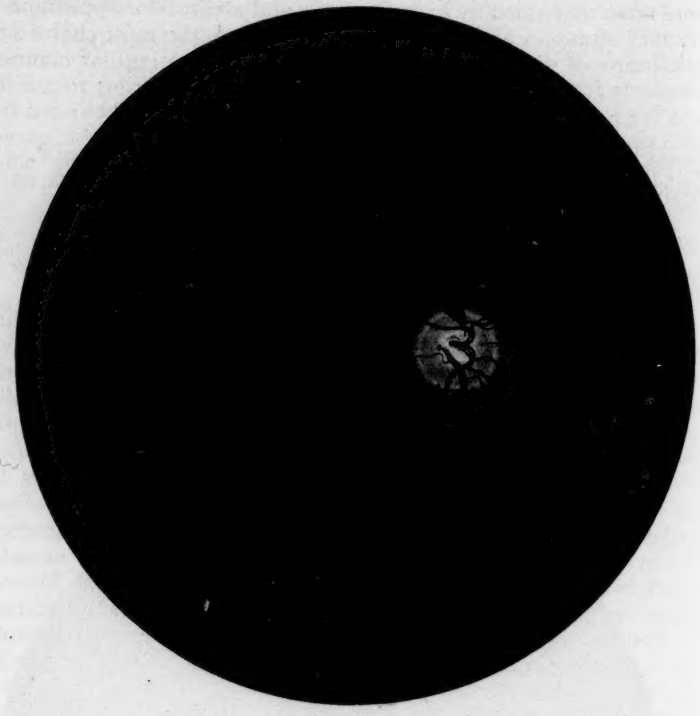


FIG. 2.

septic lesions. Hypotension was present in four and hypertension in twelve. In only seven of the cases was a mild degree of arteriosclerosis present.

(*c*) *Haemorrhages and Exudates.*

Twenty-seven cases presented a combination of the appearance of the two foregoing groups—exudates characteristic of group (*a*). looking as if they were plastered on to the retina along with the characteristic haemorrhages of type (*b*). At the time of examination the haemorrhages and exudates extended over most of the fundi. At the macula there was an incomplete ring of yellowish-white spots frequently seen. The ages ranged from 42 years to 72 years.

9—Hypotension.
5—Normal tension.
13—Hypertension.

Fifteen were under treatment with insulin, the remainder on diet. Seven had septic lesions.

(*d*) *Large Retinal Haemorrhages.*

Four patients exhibited this condition. In addition to the haemorrhages and exudates seen in type (*c*) there were copious haemorrhages, measuring sometimes to one-fourth of the length of a main temporal trunk, forming a roughly shaped quadrilateral ensheathing the vein. The bleeding was of slow onset and patients complained that everything had a reddish tinge, if the haemorrhage was large enough to come near the macula. These haemorrhages were incompletely absorbed and the vision became greatly deteriorated. The average age was 53 years. There was a septic history in every case (cellulitis and gangrene). Arterio-sclerosis of the retinal vessels was present. General blood pressure, systolic, 160-190. The tension in the retinal vessels was not taken.

(*e*) *Advanced Type.* (Fig. 3).

This was seen in fourteen patients and Fig. 3 is an example. The principal lesion consists in fairly large plaques of yellowish

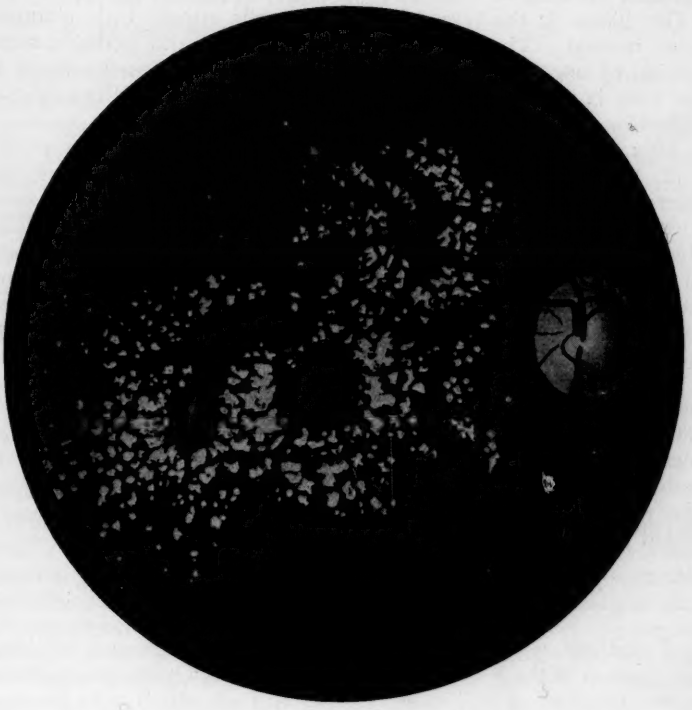


FIG. 3.

white material, formed by a coalescence of smaller patches. The macular region is chiefly affected, but the process may be seen to extend beyond the temporal vessels. The whitish-yellow areas are in close apposition to the main trunks of the temporal vessels. They appear to be an advanced degree of the first type (*a*). One or two small haemorrhages are seen among the plaques, chiefly in the macular region. This type of retinitis occurred in patients whose average age was 54 years and the duration of the diabetes was five years. Albumen was absent from the urine, but four of the cases had had septic lesions. Two cases had hypotension of the retinal arteries, two had a normal tension, and the remainder had hypertension. Eleven cases were being treated with insulin.

Condition of the Retinal Vessels
General Changes

The condition of the vessels often affords a clue, in a case of retinitis of doubtful origin, as to whether the case is one of diabetes mellitus or not.

(1) There is the type where the vessels appear little changed from normal. There is loss of translucency and perhaps some cutting of one of the lesser veins by the superimposed artery. If the vein is displaced by the artery at the crossing, the angle of deflection is small.

This type occurred most frequently in diabetic retinitis.

(2) Where there is diminution of the whole vascular trunk, the arteries are smaller, have a more rounded appearance and seem to stand out from the retina. They also seem somewhat straighter than normal. Punctate haemorrhages are frequently seen, usually at first in the peripheral parts of the retina. Fig. 2 shows this type, except that the macular region is invaded by the haemorrhages as the condition has lasted some time. Generally there is a high blood pressure.

(3) Lastly, there is the class with white spots and rather larger haemorrhages, with sometimes some oedema of the optic disc. The vessels show well-marked arterio-sclerotic changes, *e.g.*, localized narrowings of the arteries during their course on the retina. The tension in the retinal vessels is sometimes so high that only approximate readings can be taken. Though the general blood pressure is high it is frequently no higher than in Group 2.

Pressure Changes in the Retinal Arteries in Diabetes Mellitus

Technique.—The method of Bailliart was employed to obtain the tension of the retinal vessels. The Bailliart Ophthalmodynamometer consists of a piston, to one end of which the pressure-recording dial is fixed, while the other extremity has a rounded

smooth button which is placed over the tendon of the external rectus. The vessels at the disc are observed through the electric ophthalmoscope. Holding the ophthalmodynamometer in position pressure is applied till visible pulsation in the retinal arteries at the disc is noted. The first pulsation indicates the diastolic pressure; with increasing pressure a point is reached when all pulsation stops and this figure corresponds to the systolic. However, these figures do not actually represent the systolic and diastolic pressures of the central artery. It is necessary to determine the intra-ocular tension by means of a Schiötz tonometer. From this is now determined the pressure of the central artery. Bailliart and Magitot, from experiments on the cat, drew up a table showing the values of the dynamometer readings corresponding to the intra-ocular tension. The general blood pressure was estimated by a Riva Rocci apparatus, using the brachial artery.

Out of 62 cases of retinitis:

Hypotension of retinal arteries	...	...	20
Normal tension of retinal arteries	...	...	8
Hypertension of retinal arteries	...	...	34

Types of retinitis were distributed among the above groups as follows:—

Types of retinitis	Hypotension	Normal tension	Hypertension
Haemorrhages only	4	0	12
Exudates only	5	0	0
Mixed (Haems. plus exud.) ...	9	5	13
Advanced retinitis	2	2	10
Totals	20	7	35

Significance of Retinitis

(*a*) *Differentiation* from *retinitis* of *other origin.*

Much discussion has arisen as to whether there is a distinctive type of diabetic retinitis. Leber (1875) and Mackenzie (1877) stated they relied on the presence of a vitreous haemorrhage as indicative of diabetes. Hirschberg (1890), Friedenwald (1925), believe that the occurrence of white spots with fine haemorrhages in the central part of the retina is suggestive of diabetes mellitus. The absence of retinitis in the young, where diabetes is most severe, the association of a high blood pressure and frequently albuminuria, with the

condition are the principal arguments against the existence of a specific diabetic retinitis.

Cases of diabetic retinitis where albumen was absent from the urine have been reported by Mackenzie (1877), Nettleship (1886), and Juler (1892). In the present series of cases, out of sixty-six patients with retinitis, forty-nine were constantly free from albumen in the urine. High blood pressure (*i.e.*, 160 mm. Hg and upwards) occurred in 36 cases, which is fairly strong evidence that hyperpiesis is at least a factor in the production of retinitis. Lange (1929) in cases of diabetes with retinal haemorrhages found high general blood pressure with few exceptions.

(*b*) *Severity of the Diabetes.*—Eight of the sixty-six cases of retinitis came to hospital complaining of dimness of vision. In every case careful questioning elicited other symptoms, such as general weakness, loss of weight, usually for eighteen months to two years. In general, the diabetes in patients with retinitis is at first mild in type and usually controlled by diet. This insidious onset, however, is no warrant of a subsequently continued benign course. In our cases twenty-seven were under treatment by diet and thirty-nine by insulin. In seventeen of the insulin-treated patients there had been a gradual increase over two to five years in the number of units of insulin required to keep them balanced. One case had been in a pre-comatous condition through failure on the patient's part to carry out the prescribed treatment. In the remaining two cases the diabetes was progressive as shown by persistence of ketones in the urine. The majority of patients with retinitis are found among new patients. In four cases the ocular condition had, apparently, developed during treatment. A patient had been treated for sixteen years with a special diet and developed a carbuncle which necessitated administration of insulin. Eighteen months later haemorrhages began to occur in the retina. Another case developed retinitis after three years and four months' treatment with insulin, shortly after an amputation for gangrene. The third patient developed retinitis after eighteen months' treatment with insulin, while the fourth, a progressive case, complained of visual defects (due to retinitis) after one year's administration of ten units of insulin per diem. It should be added that main reliance is placed for these facts on the patients' histories. I have, however, seen patients with retinitis develop a vitreous haemorrhage while the diabetes has been under full control with diet and insulin treatment. Control of the diabetes is without effect on the retinal condition. Most of the patients with retinitis have been observed to develop fresh haemorrhages and exudates in the retina while under treatment.

(*c*) *Haemorrhages.*—Haemorrhages occurred in all but five cases of retinitis and were deeply situated, round and small, like the

imprint of a wet sponge on a wall, as Adams describes them. The origin of these haemorrhages is from the capillaries (Foster Moore). Thirty-seven of the patients with haemorrhages had a raised general blood pressure and in addition thirty-five had hypertension of the retinal vessels. In those cases with haemorrhages unaccompanied by exudate in the retina twelve out of sixteen had hypertension. Therefore, this suggests that hypertension plays a part in the production of these haemorrhages of the retina. Ellis and Marrack (1923), from a study of a series of cases of retinitis (non-diabetic), suggest that hyperpiesia is a more common cause of retinitis than chronic interstitial nephritis. On the other hand, in our cases, seven had normal pressure in their retinal vessels, and twenty-four had hypotension, and here the probable explanation is the effect of the altered condition of the blood on the endothelial cells of the vessel walls. Foster Moore (1916) found that the pressure in the intra-ocular vessels was sometimes not raised and may be less than normal. Thus in thirty-seven patients whose average general blood pressure was 138 mms. Hg the intra-ocular pressure, as measured by Schiötz tonometer, was 14.3 mm. Hg, while in other twenty-six cases whose general blood pressure was 244 mms. Hg the intra-ocular pressure was 15.3 mms. Hg.

In the present sixty-one cases of retinitis, the amount of haemorrhage varied from the small deeply seated haemorrhages to the larger type of haemorrhage designated as retinitis diabetica haemorrhagica. It has been pointed out that exudates occur after the development of multiple retinal haemorrhages (Group 2) or the primary change may be exudates without haemorrhages. These two groups tend to develop into the retinitis, most frequently seen, viz., a combination of both changes. Cases with copious haemorrhages in the retina were probably a further stage of the common form of retinitis for exudates with deep haemorrhages were also present. The general blood pressure, in these cases, ranged from 160-190 mms. Hg with marked peripheral arterio-sclerosis. This type is called haemorrhagic retinitis of diabetes.

Conclusions

1. That a characteristic form of retinitis exists in diabetes frequently and begins in the central part of the retina as whitish-yellow spots.

2. The small amount of sclerosis of the retinal vessels is an important point of differentiation from retinitis of other origin.

3. Haemorrhages may be due to the effect of toxin causing secondary changes in the vessels or to hypertension. (The latter being probably a secondary mechanical factor).

TABLE I—HYPERTENSION CASES

Sex	Age	Duration	Type of Diabetes Mellitus	Insulin or Diet	General B.P.	Retinitis Type	Retinal Arterial Pressure	A.S.	Albumen	Sepsis
F.	42	7 years	Mild	Diet and 30	160 110	Haemorrhages	60 129	Slight	—	Carbuncles
F.	57	N.K.	Mild	Diet	280 150	Haemorrhages	130*	Marked	Occasional	—
F.	49	6 years	Average	20	160 110	Haemorrhages	57	Slight	+	—
M.	61	25 years	Mild Severe	50	140 85	Advanced	70 135	Slight	—	—
F.	42	2 years	Mild	Diet	180 100	Advanced	70 135	Slight	—	—
M.	58	Several years	Mild	10	160 90	R. Haem. and exud. L. Old thrombosis	60 115	Marked	Occasional	Whitlows
F.	70	4 years	Mild	Diet	160 100	Haemorrhages	60 120	Slight	—	—
M.	62	6 years	Mild	Diet	180 100	Advanced	90 140+	Slight	—	—
F.	54	1½ years	Average	20	175 90	Advanced	60 130+	Slight	—	—
F.	57	2 years	Mild	10	160 100	Haemorrhages	50 115	Slight	+	—
F.	44	1 year	Mild	Diet	235 140	Mixed	90	—	—	—
F.	59	2 years	Moderate	Diet	250 130	Mixed	80	—	—	—

* Diastolic pressure.

* Diastolic pressure.

TABLE I—HYPERTENSION CASES—*continued*

Sex	Age	Duration	Type of Diabetes Mellitus	Insulin or Diet	General B.P.	Retinitis Type	Retinal Arterial Pressore	A.S.	Albumen	Sepsis
F.	50	1 year	Mild	Diet	195 100	Mixed	75	—	+	—
F.	68	2 years	Moderate	50	140 85	Haemorrhages	55 90	Slight	—	—
F.	63	3 years	Moderate	20	175 100	Advanced	67 110	Slight	—	—
M.	55	2 years	Severe	30	180 105	Advanced	70	Slight	—	—
F.	55	1½ years	Moderate	24	140 90	Mixed	68	Slight	+	—
F.	45	3 years	Moderate	50	140 90	Advanced	65	Slight	—	—
F.	58	1 year	Mild	Diet	180 110	Haemorrhages	65	—	+	—
M.	60	2 years	Mild	Diet	145 90	Haemorrhages	50	—	—	Threatened gangrene
M.	57	10 years	Severe	Diet	160 100	Haemorrhages	60 120	—	—	Gangrene
F.	60	1 year	Moderate	50	140 90	Advanced	47 105	Slight	—	Vaginal discharge
F.	41	5 years	Severe	72	145 90	Haemorrhages	60 125	—	—	Tonsils +
M.	61	14 years	Slight	Diet	140 90	Haemorrhages	48 110	—	+	Gangrene

TABLE I—HYPERTENSION CASES—*continued*

Sex	Age	Duration	Type of Diabetes Mellitus	Insulin or Diet	General B.P.	Retinitis Type	Retinal Arterial Pressure	A.S.	Albumen	Sepsis
F.	48	3 years	Mild	Diet	155 90	Mixed	55	Slight	—	—
F.	53	3 years	Average	Diet	160 90	Haemorrhages and spots	50	Slight	—	—
F.	68	N.K.	Mild	Diet	140 85	Advanced	70	+	—	—
F.	48	5 years	Mild Severe	Diet and 20	175 100	Advanced	65	+	—	Septic toe
F.	53	1 year	Moderate	20	200 120	Haemorrhages	90	—	—	Ulcer of foot
F.	53	3 years	Mild	Diet	120 80	Mixed	65 +	Slight	—	—
F	51	N.K.	Average	44	200 120	Mixed	70	Slight	+	Cystitis
F.	63	4 years	Mild	Diet	190 120	Mixed	65	Slight	+	—
F.	47	4 years	Average	20	160 90	Mixed	60 110	—	—	—
F.	56	3 years	Average	44	170 90	Mixed	60 130	Marked	+	Boils and carbuncles
M.	60	3 years	Average	20	160 90	Mixed	65 130	Slight	—	—

TABLE II—HYPOTENSION CASES

TABLE II—HYPOTENSION CASES

A. *Type of Retinitis=whitish yellow exudates only*

Sex	Age	Duration of D.M.	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
F.	62	2 years	Mild	Diet	130 80	20	30 85	35 90	Slight	—	—
F.	56	5 years	Moderately severe	Insulin 75 units daily	130 85	20	20 65	29 73	+	—	—
F.	40	4 years	Average	20 units	130 85	20	30 80	35 85	Slight	—	—
M.	57	1 year	Average	20 units	135 90	25	20 60	35 76	+	—	—
F.	50	2 years	Mild	Diet	135 80	20	20 70	20 75	+	—	—

B. *Type of Retinitis=Punctate Haemorrhages*

Sex	Age	Duration of D.M.	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
F.	64	1½ years	Moderately severe	52 units	190 120	15	40 115	40 100	—	—	Neuritis
F.	50	N.K.	Mild	Diet	150 90	20	30 90	35 93	—	—	—
M.	64	2 years	Mild	Diet	180 100	20	18 65	27 68	+	—	—
F.	55	4 years	Mild	Diet	190 125	25	40 100	56 110	—	+	—

TABLE II *(continued)*

C. *Type of Retinitis—Exudates and Haemorrhages*

Sex	Age	Duration of D.M.	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
M.	57	$\frac{6}{12}$ year	Mild	Diet	140 90	20	18 45	25 60	—	—	Osteo-arthritis
F.	47	3 years	Moderate	40 units	130 85	20	30 78	35 90	—	+	Gangrene
F.	42	4 years	Severe	40 units	130 85	17	25 78	27 78	—	—	—
F.	62	16 years	Moderate Severe	30 units	162 100	20	20 60	30 70	+	—	Multiple abscesses
M.	57	2 years	Severe	44 units	125 85	20	20 65	30 75	—	—	Claudication
M.	62	1 year	Mild	Diet	130 80	20	22 80	30 80	—	—	Peripheral neuritis
F.	59	1 year	Mild	Diet	220 110	20	35 80	40 85	—	—	—
M.	74	9 years	Mild	40 units	140 95	20	35 82	40 87	Slight	—	Pyorrhoea
F.	59	5 years	Moderate	52 units	135 80	20	30 72	35 80	Slight	—	Multiple abscesses

D. *Type of Retinitis—Advanced Type*

Sex	Age	Duration of D.M.	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
F.	45	2 years	Moderate	20 units	180 100	25	35 115	47 120	—	+	Cramp in legs
F.	61	6 years	Moderate	15 units	178 100	20	30 75	35 80	+	—	Coma

					100		115	120			legs
F.	61	6 years	Moderate	15 units	178 100	20	30 75	35 80	+	—	Coma

TABLE III

Normal retinal arterial pressure

A. *Type of Retinitis—Exudates and punctate haemorrhages*

Sex	Age	Duration of D.M.	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
F.	63	1½ years	Severe	40 units	170 120	20	50 110	60 110	—	—	Chr. Conjunctivitis
F.	57	3 years	Moderate	30 units	160 100	20	42 87	55 90	—	—	—
M.	57	N.K.	Mild	Diet	145 95	25	30 70	47 85	—	—	—
F.	66	8 years	Moderate	30 units	190 120	18	62 110	60 105	—	—	Claudication
M.	60	2 years	Mild	Diet	160 100	20	42 115	52 105	—	—	—

B. *Type of Retinitis—Advanced*

Sex	Age	Duration of D.M	Type of D.M.	Insulin or Diet	Brachial B.P.	Intra-ocular B.P.	Ophthalmo-dynamo-meter Reading	Retinal Arterial Pressure	A.S.	Alb.	Sepsis or other Complications
F.	58	N.K.	Mild	Diet	160 100	20	35 100	45 100	+	—	—
F.	60	1 year	Moderate	50 units	130 90	20	37 90	45 92	—	+	Leucorrhoea

Section III—The Pupillary Changes in Diabetes

The size and condition of the pupils have been the subject of much investigation in diabetic coma, in hypoglycaemia and in acidosis.

The Pupil in Coma.—Labbe (1922) states that the pupil may be contracted or dilated and possesses no diagnostic significance.

In our seven cases of coma the size and reactions of the pupil were noted in every case. In the patients admitted in coma, the pupils in three cases were contracted, and in two semi-dilated. The reaction to light was brisk in each case. Bearing in mind the varying size of the pupil in health, there seems to be no characteristic change in the size or reactions of the pupil in coma.

Hypoglycaemia.—Poos and Risse (1926) report miosis with spasm of the ciliary muscle in hypoglycaemia produced by insulin, the pupil at the height of the hypoglycaemia being half its original size. These appearances are said to be due to stimulation of the parasympathetic. According to Poos and Risse (confirmed by Jorns, 1927), this stimulation by insulin was strong enough to break down the mydriasis of atropine.

We have observed an attack of hypoglycaemia in two patients, both children. In one case there was trembling of the limbs but no actual convulsions. In the other there was pallor and nausea. In both cases the pupil showed no change in its original size, which was semi-dilated. In our other cases of hypoglycaemia some time elapsed after the attack before the patients were seen.

Acidosis.—Bourne (quoting Wieland and Schoen) states that decrease of the blood alkali, *i.e.*, by excessive respiration, causes a depression of the sphincter centre with a resultant dilatation of the pupil.

Case 1 of coma had an alkali reserve of 4.24 millimols rising to 16 millimols on treatment, the pupil, however, was semi-contracted. Further, in the cases of coma here described, no change in the size of the pupil could be found during the whole of their stay in hospital.

Conclusions

1. The size of the pupil in coma is not characteristic.
2. No change in the pupil could be observed in hypoglycaemia.

Section IV—Changes in Vitreous in Diabetes

Literature.—In 1903 Krause reported a series of cases of diabetic coma with low tension of the eye. This was confirmed by Heine, Reisman (1916), Middleton (1922), Patek (1929), Gallus (1924), however, has seen coma with no hypotension. Ascher and others say there is no accompanying fall in the general blood pressure.

Cases of Coma

1. M.28.—Had a previous attack of coma, 4½ years ago—two days before admission began vomiting.

State on admission.—Pale, dehydrated, restless—resps. 30, temp. 95°, pulse, 123—alkali reserve, 4.24 millimols; urine complete reduction of Benedict, Rothera-blue-black, a strongly positive ferric chloride test. When first examined fifteen hours later, after 153 units insulin and 320 gms. glucose, the skin had lost its elasticity. Resps. 20, temp. 97, pulse 116, the intra-ocular tension was 11 mms. Hg and the blood pressure 112 mms. Hg systolic, 80 mms. Hg diastolic. Pupils semi-contracted, reactions and fundi normal. Another estimation half-hour later gave a similar result. At this time the urine completely reduced Benedict, and gave a blue-black Rothera, and a trace of colour with ferric chloride. The alkali reserve was 16 millimols.

The patient was now put on 10 ounces saline 2-hourly alternately with 50 gms. glucose and 25 units insulin for the next 24 hours. At the end of 36 hours the intra-ocular tension rose to 15 mms. Hg and the blood pressure to 120 mms. Hg systolic and 85 diastolic. The urine had a trace of sugar. The patient was now put on carbohydrate 108 gms., protein 14 gms., fat 13 gms., and insulin 60 units with 90 gms. glucose. Twenty-four hours later the intra-ocular tension was 23 mms. Hg, the blood pressure 120 mms. systolic and 85 mms. Hg diastolic. The urine contained a trace of sugar and acetone. On the fourth and eleventh days similar readings of the intra-ocular and blood pressure were found.

2. F.37. For one month had complained of lassitude following an attack of influenza. She was very constipated.

State on admission.—In a semicomatose condition, able to drink, resps. 30, temp. 97°, pulse 88. The urine contained sugar + + + Gerhard + + + Rothera + + +. She was examined half an hour later when the intra-ocular tension was 9 mms. Hg and the blood pressure 105/75 mms. Hg.

She was given 125 units insulin and 50 gms. glucose. This patient had marked diminution of the sensitiveness of the cornea, so that it was possible to take the Schiötz reading without any cocaine. The pupils were small and reacted to light; fundi were normal.

Twelve hours later the intra-ocular and the blood pressures were the same as the first estimation. The corneal reflex was still diminished. At this time she had been given another 125 units of insulin and 250 gms. glucose, in addition to 44 ounces of water —the urine contained a trace of sugar.

Twelve hours later the intra-ocular tension had risen to 12 mms. Hg and the blood pressure to 120/90 mms. Hg. Seventy-five units insulin, 150 gms. glucose, and 58 ounces of water had been given during the day.

Twenty-four hours later the intra-ocular tension measured 12 mms. Hg, blood pressure, 125 mms. Hg.

Amount of insulin—110 units. Urine, sugar + +

Amount of glucose—200 gms.

Amount of fluid—111 ounces.

The figures for the next 24 hours were:—

Intra-ocular tension ...	23 mm. Hg	
Blood pressure	125 systolic 80 diastolic	
Insulin	90 units	Urine sugar + +
Fluid	76 ounces	Acetone +

The intra-ocular tension maintained this level when taken four and eleven days later. The acetone tended to reappear in the urine and was counteracted by the administration of glucose. The diet consisted of carbohydrate, 180; protein, 70; fat, 45; with 60 units of insulin daily.

This patient developed changes in refraction, but no difference in the intra-ocular tension could be discerned. Unfortunately she succumbed to gangrene of the lung. The case had never been treated for diabetes before the onset of coma.

3. F.8. *History.* Diabetic for two and a half years: on carbohydrate, 128; protein, 89; fat, 75 gms.; insulin, 34/34 units per diem.

Complained of severe abdominal pain and had been vomiting copiously the day before admission, during which time no insulin had been taken.

State on admission.—Very ill-looking child—marked air hunger—drowsy—able to swallow. Temp. 97.6°, pulse 108, respirations, 32.

Examined six hours later.—Pupils contracted, reactions and fundi normal, loss of corneal sensitiveness.

Intra-ocular tension	12 mms. Hg
Blood pressure	80/55 mms. Hg
Blood sugar	0.6 per cent.
Urine—Benedict + + +	Rothera + + + Gerhardt + + +

Treatment.—120 units insulin, 90 gms. glucose, fluid, 90 ounces (vomited several times).

At eighteen hours after admission.

Intra-ocular tension	15 mms. Hg
Blood pressure	90/60 mms. Hg
Blood sugar	0.75 per cent.
Urine	Sugar + + only

Treatment.—100 units insulin, 90 gms. glucose, and 29 ounces fluids.

Forty-two hours after admission.

Intra-ocular tension	20 mms. Hg
Blood pressure	90/60 mms. Hg
Blood sugar	0.18 per cent.
Urine	Sugar+

Treatment.—Fifty-two units insulin, 50 ounces fluid, plus diet of carbohydrate 150; protein, 76; fat, 30. Similar readings were obtained at subsequent examination.

4. F.24. *History.* Under treatment for diabetes for 4½ years on 40/40 insulin. For two days had suffered from diarrhoea and vomiting, during which time no insulin was taken.

State on admission.—Air hunger—acetone odour of breath; rigidity of the abdomen. Temp. 97.2°; respirations, 32; pulse, 136.

Examined six hours later.—She had been given 350 units insulin, 125 gms. of glucose, and 31 ounces of fluids.

Blood sugar equalled 0.45 per cent., urine contained much sugar with strongly positive Gerhardt and Rothera tests. Pupils semi-dilated but reacted briskly. Fundi were normal. Intra-ocular tension 85 mms. Hg; blood pressure, 85 mms. Hg (systolic) and 50 mms. Hg (diastolic). The other readings of the intra-ocular pressure, etc., were as follows:—

Eighteen hours after admission.

Intra-ocular tension...	10 mms. Hg
Blood pressure	95/65 mms. Hg
Blood sugar	0.22 per cent.
Urine—Benedict+++ Rothera++ Gerhardt++	

Treatment.—Insulin, 170 units; glucose, 155 gms.; fluid, 28 ounces.

Forty hours after admission.

Intra-ocular tension...	14 mms. Hg
Blood pressure	103/70 mms. Hg
Blood sugar	0.95 per cent.
Urine—Benedict, trace. Rothera, trace. Gerhardt, trace.	

Treatment—210 units insulin, 170 gm. glucose, plus diet, carbohydrate, 285; protein, 11; fat, 6.

Sixty-four hours after admission.

Intra-ocular tension...	18 mms. Hg
Blood pressure	110/85 mms. Hg
Blood sugar	0.76 per cent.
Urine...	Trace of sugar

Treatment.—180 units insulin, glucose, 120 gms.

During the next twenty-four hours the intra-ocular tension rose

to 23.5 mms. Hg. This tension was maintained. Patient was receiving 80 units insulin per day.

5. F.28. History. Diabetes and hyperthyroidism for five years. On 54 units insulin. The amount of insulin had been changed on several occasions as this patient suffered from hypoglycaemic attacks. She stopped her insulin and was admitted in comatose condition. Temp. 98°, pulse 130, respirations 28.

Examined six hours after admission.

Intra-ocular tension	14 mms. Hg. Pupils semi-dilated, reactions and fundi normal.
Blood pressure	105/70 mms. Hg
Blood sugar	0.4 per cent.

Urine—Benedict+++ Rothera+++ Gerhardt+++

Treatment.—110 units insulin, 90 gms. glucose, 40 ounces fluid. Twenty-four hours later the intra-ocular tension rose to 20 mms. Hg which proved by subsequent examination to be the normal level.

6. M.22. Diabetes for two years. On 40/40 units insulin. For thirty-six hours had been vomiting; continued, however, to take his insulin but no food or sugar.

Examined on admission.—Abdominal pain, marked air hunger —acetone in breath. Temp. 97°, resp. 30, pulse 130. Pupils semi-dilated, normal reactions, fundi nil.

Intra-ocular tension...	25 mms. Hg
Blood pressure	145/90 mms. Hg

Urine—Rothera+++ Gerhardt+++ Benedict++

He was given 195 units insulin and 280 gms. glucose in the first twenty-four hours, but no change could be detected in the intra-ocular pressure. This patient continued to show traces of sugar and acetone in his urine.

7. M.55. *History.* Diabetes since Nov., 1928. On 30/20 units insulin per diem. Three days before admission unable to obtain insulin. The next day he began to vomit and some time later began to feel drowsy.

Admitted in a pre-comatose condition, blood sugar 850 mg. per 100 c.c. capillary blood.

On examination seven hours after he had been given 170 units insulin and 50 gms. glucose the pupils were semi-contracted, reactions and fundi were normal. The intra-ocular tension was 22 mm. Hg and the blood pressure equalled 120 mms. Hg systolic and 88 mms. Hg diastolic. These figures were maintained on subsequent examinations.

8. M.30. *History.* Complained of great weakness and polyuria for ten weeks. On admission to hospital he had a strong smell

of acetone in his breath (blood sugar 0.29 per cent., urine, Benedict +++ Rothera+++ Gerhardt+++). Temp. 98.6°, pulse 92, resp. 20.

This patient had an intra-ocular tension of 25 mms. Hg, with a blood pressure of 115 mms. Hg (systolic) and 75 mms. Hg (diastolic). Pupils semi-dilated, reactions and fundi normal. On discharge from hospital to a sanatorium he was put on 96 units insulin daily.

Discussion

Of the above cases five were in a comatose condition, one was bordering on coma and the other patient was pre-comatose. Case 8, a severe case of untreated diabetes, is included for purposes of comparison.

The fall in Intra-ocular Tension.—The intra-ocular tension was diminished in five of the cases. The fall varied from 14 mms. Hg to 6 mms. Hg, taking the reading subsequently maintained as the normal. The time taken for the tension to reach its normal figure varied from 30 to 84 hours, depending on the amount of its initial drop.

Krause obtained a fall in intra-ocular tension in 100 per cent. of cases of coma, Heine in 21 out of 22, Weischmann and Koch (1928) in five out of seven cases of coma.

As an index of severity, Schütz found the tension varied from day to day, but at the height of the coma it was extremely low or nil. The greatest fall in tension in our cases occurred in those where there had been marked loss of fluid by vomiting and diarrhoea and in an untreated case of diabetes of approximately one month's duration. Case 1 was so markedly dehydrated that the skin had lost its elasticity.

Relation to general blood pressure.—Normally the intra-ocular pressure passively follows the general blood pressure, and this is borne out to some extent by our cases. It must be noted, however, that cases 2 and 4 had approximately the same intra-ocular tension but there was a difference of 20 mms. Hg in the blood pressure. Also, while there is a marked rise in the eye tension on recovery, the blood pressure shows a relatively smaller change.

Time of fall of eye tension in coma.—Weischmann and Koch (1928) stated that in hypoglycaemia and in some cases of cerebral haemorrhage there is a fall in the intra-ocular tension. Most observers are agreed, however, that the soft eyeball is symptomatic of diabetic coma, and it would be of interest to attempt to assess the time of its appearance in the chain of symptoms.

From a study of the histories of the above cases, the fall in ocular tension appears after the abdominal pain and vomiting. Its exact relations to the change in the respiration rate is not so

apparent and in this connection case 6 is interesting. This patient had marked abdominal respirations and yet the ocular tension was normal which suggests the development of the so-called soft eyeball follows the respiratory signs. In some of our patients it was noted that although comatose they could still swallow and this was probably automatic. Macleod has seen cases of daibetic coma "difficult to rouse, respond by automatic swallowing."

Acidosis.—The exact relationship between the fall in intra-ocular tension and the presence of aceto-acetic acid and acetone in the urine, is difficult to determine. A degree of acidosis sufficient apparently to produce general effects, *e.g.*, vomiting and increased respiratory rate, as shown by cases 6 and 7, does not cause any lowering of eye tension. At the same time it should be noted that all five cases of reduced ocular tension had much diacetic acid and acetone in the urine.

During recovery when the ocular tension was gradually assuming its normal level the aceto-acetic bodies had disappeared some time before, from the urine, except in case 5, which was complicated by hyperthyroidism. Case 2 began to have acetone in the urine after the intra-ocular tension had been normal and the acetone persisted without any change in the eye tension. Krause failed to produce lowering of ocular tension on administration of oxybutyric, diacetic acid and acetone. Dodds and Robertson (1930) found that the amount of aceto-acetic acid bears no relation to the clinical state. On the other hand, it is reasonable to assume that the amount of beta-hydroxyacetic and oxyacetic acid from the incomplete metabolism of fat, was greatest, when the patients in coma were admitted to hospital. In this connection Graham points out that aceto-acetic acid is the cause of coma and that the acetone in the urine is only a decomposition product of the diacetic acid.

Dehydration.—That loss of fluid was common to those cases which did not have reduced ocular tension, as well as to the other patients with fall in tension shows that it cannot be the sole cause. Further, Graëfe was unable to obtain a reduction in ocular tension in cholera. Lambert and Silbert (1928) believe that the decrease in tension is due to increase in the osmotic pressure of the blood and the marked dehydration of the coma cases may be the cause of the ocular change. They obtained a 40 per cent. fall in intra-ocular pressure following intravenous injection of hypertonic saline. Case 1 was the most dehydrated of all the series as shown by marked loss of skin elasticity, but he did not show such a marked fall as case 4 in whom there had been less loss of body fluid.

Hilton Fagge (1874) noted that the blood in coma is thick and flows slowly. Later there is an increase in the haemoglobin (Graham) which indicates dehydration. Graham also observed

that circulatory failure occurs in fatal cases for the blood pressure falls. This would be reflected in the ocular tension, and a disturbance of the molecular concentration in the tissues and blood is probably one of the factors in reduction of eye pressure as has been shown by Hertel (1914). The experimental work of Römer and Kochmann (1913) suggests there is present a specific tension-reducing toxin in diabetic coma, but this is considered by Poos (1930) to be due to the effect of introduction of a foreign protein in their experiments.

Conclusions

1. A reduction in intra-ocular tension occurs in a large percentage of cases of diabetic coma.
2. The fall of eye pressure occurs after vomiting and the development of respiratory disturbance.
3. The cause seems to be found in disturbed molecular concentrations of the tissue fluids and blood following the acidosis and dehydration.

Effect of Insulin on the Intra-ocular Pressure

Rises in intra-ocular tension have been described by Ascher (1925) and others. Ascher obtained a rapid temporary rise in ocular tension, the maximum occurring in thirty minutes and after two hours the tension fell. Poos (1930) found a variation of the blood sugar, in young diabetics, after administration of carbohydrate and insulin, from 650 mg. per cent. to 35 mg. per cent., while the ocular pressure varied at the same time between 13 mms. and 26 mms. Hg. In our cases, estimation of the ocular tension was made: (1) in 43 cases balanced on diet; (2) 48 cases balanced on insulin and diet; (3) 20 progressive cases; (4) 4 new cases before and after the administration of insulin; (5) three cases deteriorating on diet treatment who became balanced on the institution of insulin treatment. In all the patients included in the above groups, no case of glaucoma was seen. The estimations were made at varying times after insulin.

Of the cases treated by diet only, eight had slight hyperglycaemia, 0.2 per cent. and slightly over. The intra-ocular tension ranged from 15 mms. Hg to 25 mms. Hg in the various cases. These figures are within normal limits. No difference could thus be found in cases with hyperglycaemia. In this connection our patients who were balanced with suitable doses of insulin and suitable diet, were not sugar-free always. Occasionally from a cold, or a temporary lapse from their diet, sugar was present for a short period in the urine, but this had no effect on the intra-

ocular tension, which remained constant. The continued taking of insulin did not cause any alteration in the general level of ocular tension which was the same as in those diabetics who were being treated by diet alone.

If the intra-ocular tension is altered, either by hyperglycaemia or by insulin then changes of ocular tension should be seen in: (1) progressive cases of diabetes; (2) in new cases, before and after the administration of insulin; (3) patients who discontinue insulin treatment. In twenty patients whose diabetes was progressive, nine became balanced and in them no change in the intra-ocular tension could be found. Of the remaining eleven, four cases showed a slightly lower ocular tension than what was to be expected from the general blood pressure reading (though this is not an absolute guide). The amount of this difference, however, was too small to be decisive and on subsequent examination, during periods of balance of the general diabetic condition, did not alter. The ocular tension of the remaining progressive cases (seven) was normal. There were four new cases of diabetes and three patients who, though on diet for an average of two years, were becoming worse. Of these seven cases only two showed a rise of intra-ocular tension following insulin. Again, no change of ocular pressure could be found in patients who refused to continue their insulin. The interesting point in connection with the above cases is that, only in elderly diabetics treated at first by diet, could an alteration in the intra-ocular pressure be discerned.

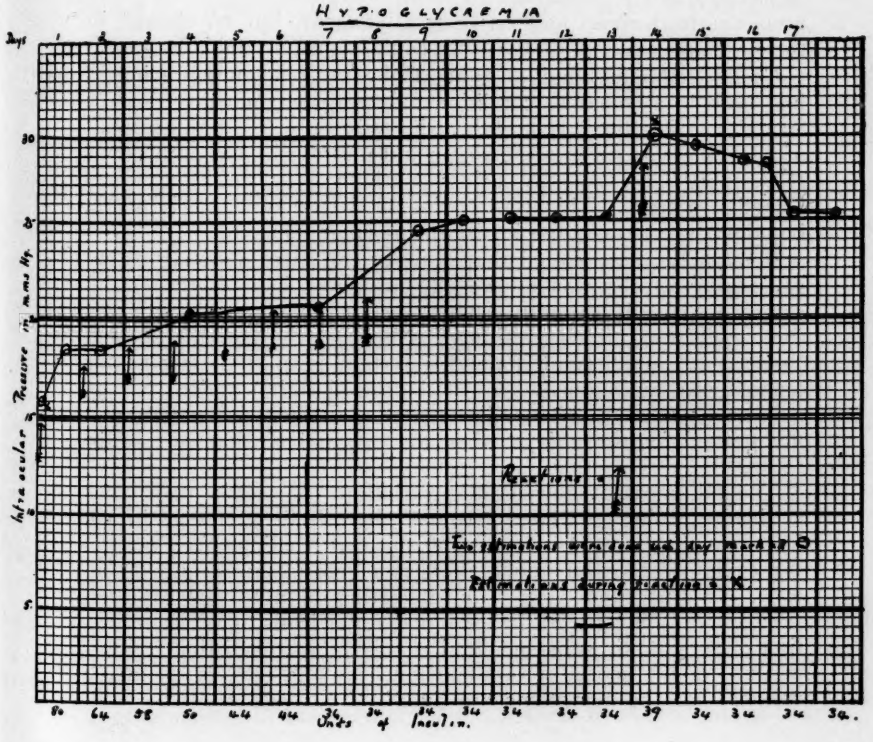
Hypoglycaemia

The effect of an overdose of insulin is to produce a condition which resembles coma and it is sometimes difficult to distinguish between the two states. Since the soft eyeball is traditionally associated with diabetic coma if it were absent in hyperinsulin coma it would be a point of considerable diagnostic importance. Unfortunately there seems to be little agreement on this point. Weischmann and Koch (1928) state that the intra-ocular tension in hypoglycaemia falls and this they believe to be due to the displacement of the body fluids; there is a rise in the haemoglobin percentage, and although there is no immediate subsequent reduction of the haemoglobin ratio this can be explained by the loss of the additional fluid through sweating which accompanies the hypoglycaemia. Weischmann and Koch estimated the ocular tension before and during hypoglycaemia in ten cases. The fall in tension which occurred in eight instances varied from 7.5 mms. Hg to 3 mms. Hg. 15 mms. Hg was the average intra-ocular tension during the hypoglycaemia. There was also a slight fall in the general blood pressure.

Eighteen cases of this series gave a history of hypoglycaemic reactions but none of them was examined till some time after the attack. This of course detracts much from the importance of the results. In six there was a slight rise in the intra-ocular tension, 2-3 mms., while in the remainder no difference could be detected.

In addition there were four cases which were examined at the following times relative to the hypoglycaemia: (1) during the attack; (2) half an hour; (3) three hours; and (4) six hours.

J.H. (aged 10 years) (see chart) showed reactions in spite of reductions in the amount of insulin given. He was examined twice daily, three and twelve hours after the morning dose of insulin. During the first estimation of his intra-ocular tension he had an attack of hypoglycaemia (pallor, sweating and felt funny). The intra-ocular tension was 15 mms. Hg. Nine hours later his tension had risen to 18 mms. Hg. For the next six days he had mild attacks of hypoglycaemia. The tension rose to 25 mms. Hg and there was little difference at the high and low



blood sugar periods of the day. The insulin meanwhile had been steadily reduced from 80 units per diem to 34 units. To check my first observation, he was given five additional units, some days after he had had no attacks. The intra-ocular tension estimated during the attack of hypoglycaemia which resulted, rose from 25 mms. Hg to 29 mms. Hg. It gradually fell to 25 mms. Hg. There was no change observed in the general blood pressure.

Another case, a child, aged 11 years, with diabetes for seven years (acute onset) was in hospital and showed reactions. In this case the intra-ocular tension curve estimated at the same time showed a gradual rise from 16 mms. Hg to 24 mms. Hg. During a period of 17 days she was examined three and a half hours after some of the attacks.

These two cases showed that with the cessation of hypoglycaemia, the intra-ocular pressure remained at a constant figure, and during the time of reduction of insulin the tension gradually rose.

Case 3.—Patient had a mild attack of hypoglycaemia at hospital, quickly remedied by administration of glucose. She was examined about half an hour afterwards, but no change in the previous reading of the intra-ocular tension could be found.

Case 4.—Patient admitted to hospital who a few hours previously had been in a comatose condition following her morning dose of 30 units of insulin. At mid-day she took only five units instead of 20. She was given some sugar and improved. On admission, B.P.—170/103, I.I.—22, urine—Rothera + + FeCl. a trace, Benedict a trace. This was obviously a case of hypoglycaemia but there was no change in the intra-ocular tension.

It must be remembered that none of the cases showing hypoglycaemia attacks was allowed to progress beyond the initial stages. A rise in the intra-ocular tension was noted in two cases (both children). This continued as long as the patients showed "reactions" and having gained a certain level, the intra-ocular tension was maintained. A small dose (5 units) added to the stabilizing amount of insulin caused a further rise which was maintained for three days when the original tension was again recorded.

The other cases showed little variation. It seems, then, that at the beginning of "insulin coma" there is little change in the intra-ocular tension. In the case of repeated reactions, *i.e.*, occurring every twenty-four hours, there is a gradual hardening of the eyes. In this connection it is interesting to note that Macleod (1930) says "that the fall in the blood sugar leads to an accumulation of some toxic substance and it may be assumed that it is on account of the action of this substance on the cells of the nerve centres that the symptoms are due."

Section V—Vitreous Haemorrhage

The occurrence of vitreous haemorrhage is especially prone to take place in diabetics with arterio-sclerosis. There were seven cases and the age period was from forty-two to sixty-one. Three of them had retinitis. The duration of the haemorrhage was on an average eleven months, and in two cases was permanent. Proliferative changes occurred, and all vision was lost. In every case the vitreous haemorrhage had occurred while the patient's diabetes was fully controlled by treatment.

Sepsis.—The most striking feature is the presence of some septic lesions in all our cases. Approximately two months elapsed between the onset of the sepsis and the vitreous haemorrhage. The history of an infected lesion suggests some connection between this and vitreous haemorrhage. Two months is a relatively long interval, however, to elapse. But this does not negative the possibility of a local endarteritis in a retinal vessel producing in course of time some aneurysmal dilatation with subsequent rupture, since the amount of bleeding suggests that it is arterial rather than venous. There is another possible explanation, that of an infected embolus, but the lapse of time between the incidence of the original local infection and that of the haemorrhage is an argument against this view.

Tension of the Retinal Arteries.—Three cases had hypertension, two hypotension and in the remainder the general blood pressure was raised. (No estimation of the retinal pressure was made in the last two cases.)

If one accepts the generally held view that the haemorrhage is due to an infected embolus lodging in one of the branches of the retinal artery causing the vessel to give way, the amount of subsequent bleeding will depend on the tension of the blood in the affected vessel and the pressure of the vitreous against the vessel wall. The pressure inside the vessel in the majority of the cases was high. Further, in the two cases with hypotension the haemorrhage must have been smaller for it had been mostly absorbed.

Case 2 is interesting in this respect for there is hypertension present, and this patient, three months after the left vitreous haemorrhage cleared, had a large haemorrhage in the right eye, which has now persisted for more than a year without any sign of absorption. Fig. 5 is a drawing of the retina of the right eye.

It has been mentioned that in the presence of healthy vitreous a considerable barrier against much extravasation would be constituted. This was shown in a case of subhyaloid haemorrhage in a young man, who was admitted to hospital a few hours after the occurrence of the haemorrhage. Several measurements of the

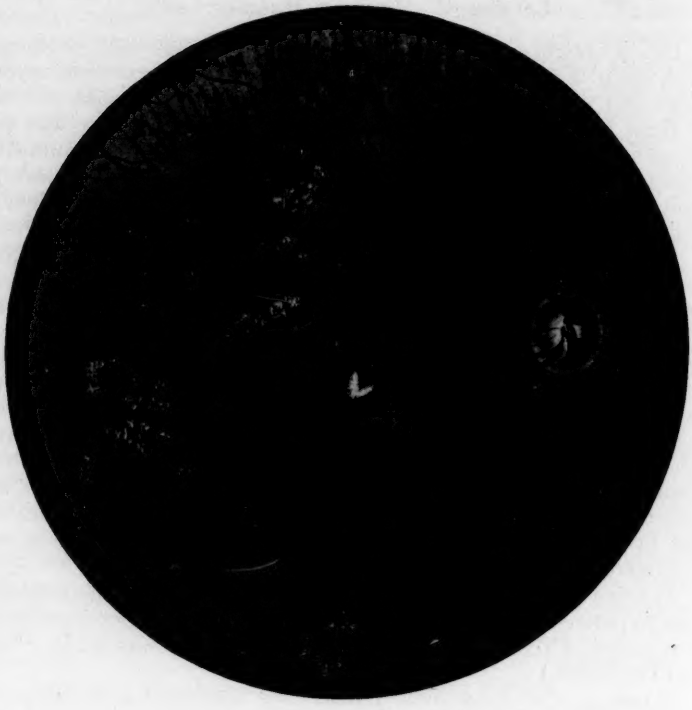


FIG. 5.

intra-ocular pressure and the tension of the retinal arteries showed these to be normal. The amount of haemorrhage was small. There is the additional factor in these cases of subhyaloid haemorrhage that the retinal vessels are apparently normal while in elderly diabetics the retinal vessels are arterio-sclerotic, and therefore less contractile from the increase in the fibrous tissue in their walls.

Conclusions

1. Vitreous haemorrhage has been found constantly associated with an infective focus somewhere in the body.

2. The amount of haemorrhage may be dependent on the state of the vitreous and the condition of the retinal vessels.

3. Hypertension in the retinal vessels is usually found in cases of vitreous haemorrhage.

VITREOUS HAEMORRHAGE

This occurred in seven cases and their histories are as follows:—

Sex	Age	Duration of D.M.	Insulin	General B.P.	Retinal Arterial Pressure	Sepsis	Lesion
F.	61	3 years	20	120 90	Rt. 25 50 L. 30 60 I. 15	Gangrene	Rt. Vitreous haemorrhages (remains)
F.	48	5 years	20	175 100	70 120 + I. 20	Septic toe	Rt. Advanced retinitis L. Vitreous haemorrhages
F.	42	3 years	30	160 95	69 125 + I. 18	Carbuncles	Rt. Haemorrhages, exudate L. Vitreous haemorrhages
M.	57	Not known	Diet	130 80	40 85 I. 20	Boils	L. Vitreous haemorrhages (remains)
F.	50	10 years	20	190 95	110 + I. 20	Carbuncles	Retinitis proliferans (Rt.)
F.	60	19 years	20	180 110	Not determined	Carbuncles	Rt. Vitreous haemorrhages L. Lens changes
M.	45	4 years	10	160 110	Not determined	Gangrene	Rt. Haemorrhages in vitreous L. Retinitis

I. Intra-ocular pressure

Section VI—Lens Changes

Visual disturbances appear early in the literature on diabetes mellitus. John Rolls (1797) in an account of two cases of diabetes writes of one patient: "there is a fullness about the eyes with a yellowish caste." Destouches (1817) noted that "quelque fois l'impression de la lumière est douloureuse; d'autres fois la vue s'affaibet. Tantôt les yeux cataractés ne permettent plus aux malades de distinguer les objets." Barendt (1834), Unger (1837) and others also established the connection between cataract and diabetes. By 1854 Mackenzie had collected a series of cases. France (1857) was the first to describe the special features of the changes in the lens in young patients which were confirmed by Warburton Begbie (1861) and hold good at the present time. Much work has been done on the causes of progressive failure of vision, *e.g.*, cataract and retinitis. Visual symptoms may, however, be transient, and recently Himsworth has stressed their importance. Thus in 49 per cent. of diabetics under 45 years and in 21 per cent. over 45 years, Himsworth obtained a history of temporary visual disturbance.

To determine the incidence of lens changes, a series of cases was divided into the following groups:—

(1) Eighty patients were examined under full mydriasis with the Gullstrand slit-lamp.

(2) In 228 patients the occurrence of macroscopic (*i.e.*, visible with the ophthalmoscope) lens changes was determined.

(3) True diabetic cataract in two cases.

At the same time special attention was paid to the duration and type of the diabetes, *i.e.*, whether the disease had progressed or remained stationary. The basis for the latter conclusion was the amount of insulin required and the absence of sugar and ketones in the urine.

By group (1) it was hoped to establish the incidence of the early changes in the lens which are commonly followed by the development of lens opacities and to determine whether diabetics were more prone than others to cataract. We have accepted the view that the only true diabetic cataract occurs in patients below 36 years old.

In the age group (23-50) there was one patient, a diabetic of four years' standing who was controlled by twenty units of insulin who had cuneiform cataract. Three had subcapsular vacuoles and in those patients the diabetes had lasted from four to seven years, one on diet treatment only and the other two on 40 and 50 units of insulin per diem, respectively. Albuminuria was present in one of these cases and the blood sugar in the others was 0.22 per cent. and 0.25 per cent.

In the age group (50-60) ten patients out of thirty-four had no

CASES EXAMINED WITH SLIT-LAMP—Age Group 23—50

Changes in lens	Vacuoles	Coronary	Cuneiform	No. lens changes
No. of cases	3	6	1	20
	4 years, blood sugar often, 0·25 per cent. diet	3 years, 15/15 insulin. Progressive	4 years, 20/20 insulin. Stationary	3 years, diet
	7 years, blood sugar 0·28 per cent, 20/20 insulin	4 years, 20/20 insulin. Progressive		2 years, diet
	4 years, 25/25 insulin. Albuminuria occasionally	4½ years, 20/20 insulin. Progressive		1 year, diet
		2 years, 10/10 insulin. Stationary		5 years, 15/15 insulin. Progressive
		1 year, diet		2 years, 20/20 insulin. Progressive
		½ year, diet		7 years, 18/18 insulin. Progressive
				3 years, 16/16 insulin. Stationary
				5 years, 35 insulin. Stationary
				2 years, 20 insulin. Stationary
				Recent, 10/10 insulin
				4 years, 15/15 insulin. Progressive
				6 years, 15/15 insulin. Stationary
				Recent, diet
				1 year, diet
				2 months B.S. ·25 per cent., 20/20 insulin. Albumen
				7 years, 20/20 insulin
				2 years, diet
				1 year, diet
				4 years, diet
				1½ years, diet

CASES EXAMINED WITH SLIT-LAMP—Age Group 50—70

	Coronary	Water Spaces	Lamellar Separation	Nuclear and Cuneiform	Nuclear	Cuneiform	No Changes
				AGE, 50—60			
No.	3	3	5	1	3	8	11
Type and Duration of Diabetes	2 years, diet, B.S. 0·33 per cent. 5 years, diet. Mild 8 years, 16/16 insulin, retinitis. Progressive	3 years, 30 units insulin. Gangrene of toe 3 years, diet, retinitis. Albumen	5 years, diet 4 years, 5/5 insulin. Progressive 7 years, 16/16 insulin. Progressive 1½ years, 12/12 insulin. Stationary 3 years, 46/45 insulin. Whitlow	4 years, 20/20 insulin. Albumen	1 year, diet 5 years, 10/10 insulin. Stationary 2 years, 30/35 insulin. Progressive	1 year, 10/10 insulin 3 years, diet. Retinitis 7/12 years, diet. Retinitis 2 years, diet. 2 years, B.S. 0·26 per cent., albumen. Diet 7 years, 12/12 insulin. Progressive 3 years, 10/10 insulin. Retinitis 10 years, 16/16 insulin. Stationary	1 year, diet. Retinitis 2 years, diet 1 year, diet 7 years, diet 1 year, 15/15 insulin. Ketonuria 3 years, 10/10 insulin 1 year, 5/5 insulin 1 year, diet 3 years, diet 4 years, 10/10 insulin 1 year, diet
				AGE, 60—70			
No.		3	3	1	2	4	3
Type and Duration of Diabetes		2 years, diet 2 years, diet. Left cuneiform. B.S. 0·25 per cent. 5 years, 40/40 insulin. Progressive	4 years, diet 5 years, diet 3 years, 45/45 insulin. Whitlow	3 years, diet. Albumen	7 years, 40/40 insulin 5 years, 20/20 insulin. Gangrene of toe	1½ years, diet, B.S. 0·3 per cent. Leucorrhoea 4 years, 10/10 insulin 6/12 years, diet, B.S. 0·33 per cent. 8 years, 10/10 insulin. Albumen	2 years, diet 4 years, 10/10 insulin 2 years, 20/25 insulin

LENS CHANGES PRODUCING OPHTHALMOSCOPIC SIGNS

	Cuneiform	Nuclear and Cuneiform	Nuclear	Lens Extractions	Complicated
			AGE, 50—60		
No.	10	6	4	1	1
Type and Duration of Diabetes	2 years, diet (age 48) 5 years, retinitis. diet 2 years, 30/30 insulin. Retinitis. 1 year, 10/10 insulin 13 years, diet 3 years, 35/35 insulin 20 years, 10/10 insulin 18 months, 35/35 insulin 15 years, diet, B.S. 308 per cent. Retinitis 4 years, 10/10 insulin	5 years, diet 5 years, diet, B.S. 0·3 per cent. 6 years, diet 3 years, diet 1 year, diet 9 years, diet. Albumen	2 years, diet 5 years, 15/15 insulin 1 year, 22/25 insulin 2 years, diet (age 48)	2 years, diet. Ulcer of foot (age 47)	1 year, diet
			AGE, 60—70		
No.	2	4	2	1	1
Type and Duration of Diabetes	2 years, diet, B.S. 0·3 per cent. 3 years, diet	2 years, 5/5 insulin 8 years, diet 2 years, 20/20 insulin. Progressive 4 years, diet, B.S. 0·35 per cent.	3 years, diet 8 years, diet	3 years, diet	8 years, diet. Ulcer of foot

changes in the lens. In fifteen there were various forms of cataract while eight had presenile changes. In the next age period (60-70) three patients had clear lenses, seven had cataract, three had lamellar separation and three other patients had fluid spaces (*Wasserspalten*). Out of 228 diabetics there were thirty cases of lens changes visible with the ophthalmoscope. Half of the patients in the age group (50-60) with cuneiform striae did not complain of visual disturbances. There were two complicated cataracts and two extractions with good resulting vision.

Cases with true Diabetic Cataract

Mackenzie (1854) wrote of the occurrence of cataract in women from 18-25 years old with diabetes and in males at a later age period. France (1857) points out that rapidly increasing bilateral opacity of the lens was characteristic of diabetes. These cases give a characteristic appearance of small subcapsular opacities, and have been fully described by Goulden, Schyder and others. The patients are young and suffer from a fairly severe form of diabetes.

In the present series there were two such cases (1) male (26), diabetic for five years, requiring 20/20 units of insulin daily, who sometimes had a trace of sugar in the urine; (2) female (17), whose sight first became affected two years after the onset of diabetes. The blood sugar was 75 mgs. per 100 c.c. of capillary blood.

Age.—In attempting to determine the effect of diabetes in producing lens changes, the influence of age must first be assessed. This has already been investigated by Anderson (1924) and Gradle (1926) who compared the incidence of lens changes in diabetics with healthy patients. Anderson's figures for these two groups of patients show little difference. When compared with the incidence of presenile and senile cataract ratios of Anderson, Schild or others, our diabetic cases have a higher incidence of lens changes. Thus:—

Age period 23-50, twenty had clear lenses while ten had presenile and senile changes—33.3 per cent. Between 50-60 years of age, eleven out of 34 cases had clear lenses, while 23 had changes —67.6 per cent. Thirteen cases out of sixteen at age period 60-70 years had lens changes—81 per cent. These figures are higher than those of Anderson and Gradle, but it must be remembered they include presenile changes as well.

The increase proportional to age shows that senile processes play a considerable but not the whole part in the production of lens opacities in diabetics.

Type and Duration of the Diabetes.—An examination of the table (age 28-50) shows that, considered as a whole, the patients with lens changes had a more severe form of diabetes than those in whom no lens change could be detected. There was a tendency to the maintenance of a higher level in the blood sugar and in others the amount of insulin was relatively higher. This is also true of the table (age period, 50-60) and (60-70). Thus, out of five cases with lamellar separation, three were definitely progressive and severe. Parsons (1904) believes that diabetic cataract should be included in the group of toxic cataracts due to nutritional defect by the toxic metabolic products. He points out that ordinary senile cataract may occur in diabetics and diabetic cataract may not differ from the senile form. There have been many investigations into the frequency of cataract in diabetes and the figures vary up to 25 per cent. (von Graefe). Baldwin and Barthel (1924) suggest the asssociation between cataract and hyperglycaemia. It has been found, however, that the amount of sugar in the aqueous is much smaller than that required to produce lens changes experimentally. Hyperglycaemia was present in eight of our cases out of 80 cases examined with the slit-lamp, which had lens opacities, and in four out of 30 patients with macroscopic cataract.

The Influence of Infections.—A history of infection was of fairly common occurrence in diabetics with cataract. One quarter of the number of patients with gangrene had lens opacities. In those cases examined with the slit-lamp there were five which had septic lesions, while a number of others had albumen sometimes present in the urine. Leyton (1927) has drawn attention to the upset of metabolism in infection, causing the blood sugar to rise, and that the factors whether insulin will be efficacious or no are not known. It seems that an increase either in the blood sugar or in toxic substances from the disturbance of metabolism may play some part in the production of lens changes.

Retinitis and Cataract.—Gallus was of opinion that the two were not associated. Anderson in thirty-two cases of retinitis found slight lens changes. In seven instances in the present investigation lens opacities and retinitis were found together.

Insulin and Cataract.—The effect of insulin treatment, at present, can be judged from two aspects only: (1) the effect on true diabetic cataract; (2) as an adjunct to pre-operative treatment.

Nettleship and Seeger (1870) reported cases where the lens opacities in young patients cleared during the course of medical treatment. On the other hand, Foster Moore (1925) reported a case in whom after five months of complete control by insulin no change could be detected. He adds that insulin may retard the progress of the lens changes. Case (2) of group (3) was under careful treatment for two years yet the cataract progressed, probably

much more slowly than it would have done on diet treatment alone.

Insulin and Operative Treatment.—In the pre-insulin days suppuration often ensued after operation especially in young diabetics. France (1860) reports such a case. In 1860 Swain reported a successful operation in a woman, aged 39 years. Gifford (1911) estimated that in 43 per cent. of young diabetics the eye was lost from suppuration, while in the old the prognosis was much better and the chief danger was retinal haemorrhage. Whiting (1914) concludes that there is little more danger than with senile cataract provided careful pre-operative treatment has been carried out.

The value of insulin in ophthalmic as well as in other operations was early recognized and now the prognosis of cataract needlings and extractions in diabetics is as favourable as in healthy persons.

Section VII—Ocular Nerve Paresis in Diabetes

Ocular palsies have been described occurring during diabetes by a large number of observers, Ogle (1866), Haltenhoff (1873), and others.

The muscles affected are the sphincter pupillae, levator palpebrae superioris (Seeger) and others supplied by the third cranial nerve (Ogle, 1866). The sixth cranial nerve (Cohn, 1878), Hawthorne (1899), is also frequently affected. Dieulafoy (1905) states that the abducens is more often involved than any other and that it is sometimes the only sign of diabetes in adults. So that a sixth nerve lesion should make one think of diabetes after excluding parasyphilitic lesions. Sometimes the fourth nerve is affected (Kwiatowski).

The third nerve was affected in two of our cases of diabetes, (both women). They had been treated for four and eight years respectively for diabetes, one by diet and the other had been for two years on insulin. Their ages were 60 and 63 years, both had arterio-sclerosis, and one pronounced diabetic retinitis. Symptoms were sudden in onest and consisted of unilateral ptosis with external strabismus. After two to three months on iodides recovery was complete.

An ophthalmoscopic examination in diabetes mellitus is helpful in assessing the general condition of the patient; in detecting the presence of arterio-sclerosis and in the diagnosis of coma and its degree. In spite of careful treatment, retinitis usually advances and is a frequent cause of failure of vision, while the danger of vitreous haemorrhage is ever present. There seems to be some

relation between the progressive deterioration of vision (cataract and retinal haemorrhages) and the presence of sepsis probably from the ensuing progression of the diabetes.

I am indebted to Professor C. C. Okell for rearranging the text greatly to its advantage. To Professor Elliott, of University College Hospital; Dr. George Graham, of St. Bartholomew's Hospital; Dr. Leyton and Professor Ellis, of the London Hospital for permission to examine their cases. To Mr. Goulden for his kindly interest and for the loan of his slit-lamp; to Miss R. M. Simmonds for her help especially the use of her card index of patients.

REFERENCES

ARTERIO-SCLEROSIS

Grafe, E., von Noordon, C. and Isaacs, S.—Die Zuckerkrankheit und Ihre Behandlung. Achte Auflage, p. 325, Berlin, 1927.
Gunn, M.—*Trans. Ophthal. Soc. U.K.*, Vol. XVII, p. 361, 1898.
Joslin, E.—The Treatment of Diabetes Mellitus. 4th Ed., Philadelphia, 1928.
Moore, R. Foster.—Medical Ophthalmology. 2nd. Ed., London, 1925. *Quart. Med. Jl.*, Vol. X, p. 29, 1916-1917.
Spalding and Curtis.—*Boston Med. and Surg. Jl.*, Vol. CXCVII, p. 165, 1927.

RETINITIS

Bailliart, P.—La circulation retinienne a l'état normal et pathologique. Paris, 1923.
Desmarres.—Traité Theor. des maladies des Yeux. 2nd Ed., p. 521, Paris, 1858.
Ellis, A. and Marrack, J.—*Lancet*, Vol. I, p. 893, 1923.
Moore, R. F.—*Loc. cit.*
Haltenhoff, G.—*Zehender's Klin. Monats. f. Augenheilk.*, Vol. XI, p. 291, 1873.
Juler, H.—*Trans. Ophthal. Soc. U.K.*, Vol. XII, p. 138, 1892.
Lange, F.—*Arch. of Ophthal.*, Vol. II, p. 551, 1929.
La Russo, D.—*Ann. di Ottal.*, Vol. LV, p. 222, 1927.
Leber, Th.—*Arch. f. Ophthal.*, Bd. XXI, Abth. 3, p. 226, 1875.
Mackenzie, S.—*Roy. Lond. Ophthal. Hosp. Rpts.*, Vol. IX, p. 134, 1877.
Moore, R. F.—*Trans. Ophthal. Soc. U.K.*, Vol. XXXVI, p. 319, 1916.
Nettleship, E.—*Trans. Ophthal. Soc. U.K.*, Vol. VI, p. 332, 1886.
Noyes, H. D.—*Trans. Amer. Ophthal. Soc.*, Vol. I, p. 71, 1869.
Parsons, J. H.—Pathology of the Eye. Vol. IV, p. 1308, London, 1904.

PUPILLARY CHANGES

Francis and Koenig.—*Trans. Amer. Med. Assoc.*, Vol. LXXXVII, p. 1373, 1926.
Jorns, G.—*Zeit. f. d. ges. Exp. Med.*, Vol. LIV, p. 179, 1927.
Labbé, M.—A Clinical Treatise on Diabetes Mellitus. English Ed., London, 1922.
Poos, F. and Rissi, O.—*Arch. f. Exp. Path. u. Pharm.*, Vol. CXII, p. 176, 1926.
Wieland, H. and Schoen, R.—*Arch. f. Exp. Path. u. Pharm.*, Vol. C, pts. 3 and 4, 1923.

OCULAR TENSION

Ascher, K. W.—*Berichte der Deuts. Ophthal. Gesell.*, Vol. XLV, p. 186, 1925.
Dodds, E. C. and Robertson, J. D.—*Lancet*, Vol. I, p. 852, 1930.
Fagge, Hilton.—*Guy's Hosp. Rpts.*, Vol. XIX, p. 130, 1874.
Gallus, E. (Bonn).—*Klin. Monatsbl. f. Augenheilk.*, Vol. LXXIII, p. 491, 1924.

Graefe, A.—*Arch. f. Ophthal.*, Vol. XII, p. 211, 1886.
Graham, G.—Pathology and treatment of Diabetes Mellitus. Oxford, 1926. *St. Barts. Hosp. Rpts.*, Vol. LXV, p. 55, 1929.
Heine, L.—*Klin. Monatsbl. f. Augenheilk.*, Bd. LXIV, Teil 2, p. 451, 1906. Berichte ueber die wl. *Versamm. d'Ophthal. Ges. Heidl.*, Vol. XXIX, p. 273, 1903.
Hertel, E.—*Arch. f. Ophthal.*, Vol. XC, p. 314, 1915.
Krause, P.—*Verhand. d. Kon. Inn. Med.*, Vol, XXI, p. 439, 1904.
Joslin, E. P.—Treatment of Diabetes Mellitus. 4th Ed., Phil., 1928.
Lambert, R. and Silbert, S.—*S. Jn. Amer. Med. Assoc.*, Vol. XC, p. 1435, 1928.
Macleod, J. J. R.—Physiology and Biochemistry in Modern Medicine. 6th Ed., London, p. 917, 1930.
Macleod, J. J. R. and Campbell, W. R.—Insulin, its use in the Treatment of Diabetes. London, 1925.
Middleton, W. S.—*Wisconsin Med. Jl.*, Vol. XXI, p. 458, 1922.
Patek, A.—*Jl. Amer. Med. Assoc.*, Vol. XCII, p, 438, 1929.
Poos.—*Klin. Monatsbl. f. Augenheilk.*, Vol. LXXXIV, p. 340, 1930.
Reisman, D.—*Jl. Amer. Med. Assoc.*, Vol. LXVI, p. 85, 1916.
Römer.—*Klin. Monatsbl. f. Augenheilk.*, Vol. LI, p. 592, 1913.
Schütz, L. G.—*Inaug. Dissert.*, Strasburg, 1913.
Weischmann, E. and Koch, F.—*Münch. med. Wochenschr.*, Vol. LXXV, p. 1160, 1928.

CATARACT

Anderson, C. L.—*Acta Ophthal.*, Vol. II, Fasc. 3, p. 250, 1924.
Baldwin, H. and Barthel, E.—*Jl. Amer. Med. Assoc.*, Vol. LXXXIII, p. 994, 1924.
Begbie, Warburton.—*Edin. Med. Jl.*, Vol. VI, p. 1105, 1861.
Berendt, F. A. G.—*Vevgl. Klin. Mittheit.*, Greifswalde, 1834.
Destouches, A. A.—"Du Diabette sucre." Thèse de Paris, p. 24, 1817.
France, J. F.—*Roy. Lond. Ophthal. Hosp. Rpts.*, Vol. I, p. 273, 1858.
Gallus, E.—*Zeitschr. f. Augenheilk.*, Vol. XLVIII, p. 89, 1922.
Gifford, H.—*Ophthal. Record*, Vol. XX, p. 243, Chicago, 1911.
Goulden, C. B.—*Trans. Ophthal. Soc. U.K.*, Vol. XLVIII, p. 104, 1928.
Gradle, H. S.—Jackson Birthday Book, Wisconsin, p. 255, 1926.
Graefe, A.—*Arch. f. Ophthal.*, Vol. IV, 2, p. 230, 1858.
Himsworth, H. P.—*Brit. Med. Jl.*, Vol. II, p. 1184, 1932.
Leyton, O.—Treatment of Diabetes Mellitus. 4th Ed., London, 1927.
Mackenzie, W.—Diseases of the Eye. 4th Ed., London, 1854.
Moore, R. F.—*Loc. cit.*
Nettleship, E.—*Trans. Ophthal. Soc. U.K.*, Vol. CIX, 1885.
Parsons, J. H.—*Loc. cit.*
Unger.—*Zeitschr. f. Ophthal.*, 1836.
Rollo, J.—Account of two cases of Diabetes Mellitus, London, 1797.
Schynder, W. F.—*Klin. Monats. f. Augenheilk.*, Vol. LXX, p. 70, 1923.
Siegen, J.—Der Diabetes Mellitus. Vol. CVII, Leipsig, 1870.
Swain, W. P.—*Roy. Lond. Ophthal. Hosp. Rpts.*, Vol. III, p. 331, 1860-1.
Whiting, M.—*Practitioner*, Vol. XCII, p. 573, 1914.

OCULAR PALSIES

Cohn, H.—*Arch. f. Augenheilk. u. Ohrenheilk.*, Vol. VII, p. 33, Wiesbaden, 1878.
Dieulafoy.—La Presse médicale. Pp. 713-716, Paris, 1905.
Hawthorne, C. O.—*Lancet*, Vol. II, p. 876, 1899.
Kwiatowski, W.—Etude general sur les affections oculaire diabetique. Thèse de Paris, No. 179, 1879.
Ogle, J. W.—*St. George's Hosp. Rpts.*, Vol. I, p. 157, London, 1866.

ANNOTATION

The Wrong Drop

It is not unusual to hear at times during the course of one's professional career of cases in which the wrong drop has been instilled into the eye with disastrous results. In earlier days when the teaching of ophthalmology during the student's curriculum was not made so much of as it is nowadays, many people on and after qualification seemed to regard atropine as a panacea for all ills of the eye; and most of us must have seen cases of primary glaucoma which have had atropine instilled, under the mistaken notion that the disease was iritis. The converse of giving eserine in cases of iritis does not appear to be so common. It has long been known that injudicious application of silver nitrate of excessive strength to the eyes in cases of ophthalmia neonatorum may result in as disastrous results as if the purulent conjunctivitis had not been treated at all. A recent case at the Birkenhead Infirmary, where a new born infant had nitric acid applied to the eyes shortly after birth, has resulted in blindness and was brought before the Liverpool Assizes on June 21. The child's father claimed damages against the Birkenhead Corporation; the medical superintendent of the Infirmary and the nurse who applied the drops. The case was settled after consultation in Mr. Justice Atkinson's room. The Corporation have undertaken to send the baby to a sunshine home, to be responsible for its education and later for its training for a blind profession. The case against the medical superintendent could not stand as the doctor was away on holiday when the accident occurred. The action against the nurse was discontinued and no order against her for costs was to be made.

It is always easy to be wise after the event and we have nothing but pity to offer to the nurse who made the mistake, which is after all perfectly understandable. But we contend that the instillation of drops of nitrate of silver into the eyes of new-born babies should always be performed by the medical man in attendance. The mistake is likely to arise because nitric acid is one of the stock reagents for the testing of urine.

The Prevention of Blindness Committee recommended that midwives should not be allowed to instil drops of any kind into the eyes of the new born and that nitrate of silver should only be used under the immediate supervision of a medical man. The midwife may be expected to test the urine of the mother and this is how nitric acid happens to be among the solutions in the midwife's bag. Her certificate allowing her to attend labour cases contains a clause, making it obligatory on her to notify the least

suspicion of discharge from the eyes of a new-born baby. In this way the authorities are enabled to see that such cases have proper treatment by a medical practitioner at an early date; and so there is no real need for the midwife to do more to the baby's eyes than clean them thoroughly with warm water.

THE INTERNATIONAL ORGANIZATION OF THE FIGHT AGAINST TRACHOMA

Assembly of Delegates; April 18, 1933, at Madrid

Professor Dr. Emile de Grósz, the President of the Organization, in addressing the assembly of delegates spoke of the work done in combating trachoma since the first International Ophthalmological Congress held in Bruxelles in 1857. He praised the advances in prophylaxis and commented on the fact that up to date the aetiology of trachoma still eluded the investigator. Also he stated that little or no progress had been made in the treatment of this disease and recourse had to be made to old empirical therapeutic methods.

The programme of the Organization was defined at Geneva in 1930, but the execution of this has been seriously embarrassed by the economic depression of the world. At a meeting in Paris in 1931, the executive committee decided that papers on the aetiology of trachoma should be read at the Fourteenth International Ophthalmological Congress at Madrid, and these were contributed by Messrs. MacCallan, Morax, Angelucci, Pittaluge and Thygesson. Dr. F. Wibaut read a paper on the initial symptoms of trachoma. Professor Dr. Emile de Grósz commented on the work done by the joint winners of the Royal Hungarian Government's prize for the best work on the cause of trachoma.

He expressed the hope that this international organization against trachoma would grow and become powerful enough to make its influence felt in a universal and practical manner.

It was decided to hold at the next meeting of the organization a discussion on prophylaxis and international co-operation. The executive committee will hold a meeting at Budapest in June, 1934, and a plenary meeting of delegates is planned for 1935, either in Holland or Switzerland.

ABSTRACTS

I.—CONJUNCTIVA

(1) **Lehrfeld, Louis (Philadelphia). — Vernal conjunctivitis.** *Arch. of Ophthal.*, September, 1932.

(1) **Lehrfeld's** comprehensive article is based on observations he has made at the Wills Hospital on 87 cases of spring catarrh seen during the years 1929, 1930, and 1931. One of his outstanding conclusions is that many cases are unrecognized, the palpebral variety being regarded as follicular conjunctivitis and the limbal form as phlyctenular disease. The two are separate entities though due to the same cause, which is probably allergic sensitiveness to street dusts, animal dusts or pollens. Males are affected more frequently than females, the proportion in the present series being 62 to 25. Forty-six cases were of the palpebral and 41 of the limbic type. In conjunctival smears from 69 cases, 40 showed eosinophils and in blood counts on 72 cases, 31 revealed an eosinophilia of over 4 per cent. The oldest patient was aged 59 years and the youngest two years. The limbic form has the following characteristics: it is commoner in children, leaves no permanent changes, or ulcers, and involves both eyes. Single discrete, or crops of vesicles may appear round the cornea, most commonly at the upper margin and they may become confluent. Irritation is usually only slight. The palpebral forms manifest three varieties: (1) Simple follicular, characterized by itching and redness with only slight pathological changes, the so-called milky film not appearing until after two or more occasional attacks. (2) The cobblestone type, seen after the disease has lasted two or three seasons and characterized by the formation of large granulations. When well established, a characteristic discharge of thick ropy lardaceous material may be peeled off from the conjunctival surface of the upper lid, without any bleeding. This discharge is likened by the author to chewing gum. (3) The giant form, where large granulomata with deep fissures between them occupy the entire conjunctival surface of the upper lids. It is remarkable that the palpebral form of the disease does not occur in negroes.

In an endeavour to find the cause of vernal conjunctivitis, the author used a series of skin tests with various pollens, proteins and dusts. The scratch method is practically useless, only three out of 23 patients giving positive reactions, whereas the intradermal injection in nine cases gave positive results in all with one or other of the test substances used. An interesting proof of the allergic nature of spring catarrh was afforded by introducing into

the conjunctival sacs of two patients substances to which they were sensitive. Although this was done in February, quite an intense reaction was produced, with irritation and lacrymation.

The author's opinion of radium is that "it is not a cure-all for vernal conjunctivitis, but that it is valuable in alleviating the itching in all forms and in reducing the pathological changes in the advanced chronic cases of the cobblestone type."

With regard to treatment, the best results are obtained by repeated cleansing of the conjunctival sacs with cold boric lotion. This should be done at least seven times a day. When there are large granulations the following drops should be instilled three times daily for the first week:

Phenocaine gr. $\overline{iv}$
Epinephrine (1:1000) m. $\overline{xx}$
Aq. Dest. ad ℥j

F. A. W-N.

(2) **Pascheff, C. (Sofia).—Researches on the diffuse lympho-cellular (lymphomatous) hyperplasias of the conjunctiva. (Recherches sur les hyperplasies lympho-cellulaires (lymphomateuses) diffuses de la conjonctive).** *Arch. d'Ophtal.*, August, 1932.

(2) **Pascheff** has already published reports of his investigations, extending over many years, in diseases of the conjunctiva, and in a paper in 1930, suggested a classification of the hyperplasias of this structure. The present communication which is in some respects supplementary to his former writings, deals with the lympho-cellular or lymphomatous varieties. These he divides into four groups with the following titles:—

1. Diffuse leucaemic hyperplastic conjunctivitis.
2. Diffuse hyperplastic conjunctivitis of orbital origin.
3. Diffuse hyperplastic conjunctivitis, metastatic and symmetrical, secondary to lymphomata of the skin.
4. Diffuse simple hyperplastic conjunctivitis.

In each group examples with full clinical and pathological reports are given.

The length of this paper and the exceptional amount of detail are such that brief excerpts therefrom would convey little to the reader and be unfair to the writer. The paper is illustrated by numerous photographs of the clinical and anatomical appearances, and is followed by a bibliography of more than usual merit, by reason of the scattered distribution of the literature of this subject.

J. B. LAWFORD.

(3) **Jordan, S. (Hamburg).—Contribution to tuberculosis of the conjunctiva. (Ein Beitrag zur Bindehauttuberkulose).** *Zeitschr. f. Augenheilk.*, Vol. LXXVI, p. 147, 1932.

(3) **Jordan** records two cases illustrative of tuberculosis of the conjunctiva. In the first a yellowish nodule, the size of a pea, was removed from near the limbus in a girl, aged 15 years. The tuberculous nature of the lesion was only recognized histologically. Subsequent radiological examination revealed an apparently simultaneously developed pulmonary lesion; the conjunctival lesion is held to have been of endogenous origin. In the second case, recurrent acutely inflamed conjunctival tissue at the lower fornix was excised, as there was no response to local treatment. Histologically no tuberculous evidence was found and animal inoculation was negative. But as further recurrences took place and in view of evidence of healed tuberculosis, as shown by scarring in the neck and by X-ray findings in the lungs, the patient was treated with tuberculin. The result was striking on this and subsequent occasions when further recurrences took place.

ARNOLD SORSBY.

(4) **Longchampt, J. and Reboul, J. (Toulon).—Four cases of Parinaud's conjunctivitis. (Quatre cas de Conjonctivite de Parinaud).** *Arch. d'Ophtal.*, February, 1932.

(4) **Longchampt** and **Reboul** record an unusual experience: four examples of Parinaud's conjunctivitis came under their observation within a month. The cases all occurred in persons who were in immediate and daily contact with animals. Two of the patients belonged to a family whose occupation was the care of sick poultry; one was a farmer, one a midwife who had been a visitor on a farm for several weeks.

In the two relatives, mother and daughter, while the ocular manifestations were slight, the constitutional symptoms were severe and prolonged.

J. B. LAWFORD.

(5) **Taborisky, J. (Haifa).—The Provaczek-Halberstädt bodies and their clinical importance. (Die Provaczek-Halberstäderschen Körperchen und ihre klinische Bedeutung).** *Arch. f. Ophthal.*, Vol. CXXIV, p. 455.

(5) An enquiry into the connection between the presence or absence of Provaczek-Halberstädt bodies and the clinical picture of trachoma and other forms of conjunctivitis led **Taborisky** to the conclusion that the presence of these bodies practically always indicates trachoma, or as he shortly puts it "without inclusions, no trachoma."

Cases of conjunctivitis with inclusion bodies (apart from inclusion blennorrhoea) are extremely rare, and in their initial stage always closely resemble acute trachoma, if they are not identical with it; this shows the Provaczek-Halberstädt bodies are not an accidental phenomenon, but are the cause of their clinical symptoms. These bodies in such cases are of a non-virulent type and probably of genital origin.

The specific significance of the Provaczek-Halberstädt bodies in trachoma is demonstrated by the striking parallelism between their appearance, development, disappearance, and the clinical features and course of trachoma.

These bodies behave differently in different parts of the conjunctiva according to the resistance of the epithelial covering to their development: the greatest resistance is offered by the corneal epithelium, followed by that at the limbus, the conjunctiva sclerae and the least in the palpebral portion.

The diagnostic value of the Provaczek-Halberstädt bodies is specially important in the differential diagnosis between trachoma and similar varieties of conjunctivitis.

Inclusion blennorrhoea is a form of conjunctivitis resembling trachoma, due to a genital variety of the Provaczek-Halberstädt bodies. Chronic blennorrhoea is most probably a mixed infection of gonococci and other micro-organisms with Provaczek- Halberstädt bodies, and in this condition the papillary hypertrophy with its recesses forms a favourable nidus for their growth.

THOS. SNOWBALL.

II.—TRACHOMA

(1) **Schousboe (Algeria).—The clinical diagnosis of the initial stage of trachoma, with graphs. (Étude des aspects cliniques du debut du trachome a l'aide de graphiques).** *Rev. Internat. du Trach.*, January, 1933.

(1) Since microscopical, bacteriological, and serological investigations have proved to be without value in the diagnosis of trachoma, up to the present time, we must rely on clinical observation. **Schousboe** gives a good drawing of early trachomatous pannus associated with corneal trachoma follicles *in esse* and after cicatrisation as portrayed by Herbert in the *Transactions of the Ophthalmological Society of the United Kingdom,* Vol. XXIV, p. 67, 1904. These are now known as the rosettes and peripheral pits of Herbert. The broadcast warnings in European and Eastern medical journals which have appeared during the last few years

should have prevented the author from attributing this important clinical observation to a recent writer.

A. F. MACCALLAN.

(2) **Wilson, Rowland (Cairo).—The corneal vessels in Egyptian trachoma.** Sixth Annual Report of The Giza Memorial Ophthalmic Laboratory. 1931.

(2) Trachoma in Egypt usually commences in the first year of life and **Wilson** has never seen a year-old child with trachoma who did not show evidence of new vessel formation in the cornea. Further he has had opportunity of examining many infants who have had definite evidences of trachoma for only a month or two, and all have shown definite new vessel formation in the cornea. An examination with the slit-lamp has shown that vascular changes in the cornea may be expected almost immediately after the appearance of definite lesions in the tarsal conjunctiva. This bears out the previously expressed opinion of the reviewer that great hesitation in diagnosing trachoma should be observed in any case in which there are no free vessels running into the clear corneal tissue from the normal or elongated vascular loops.

A. F. MACCALLAN.

(3) **Busacca (Brazil).—The frequency of corneal complications in trachoma deduced from the examination of a hundred cases. (La frequenza delle complicazione corneali nel tracoma dedotta dallo esame di cento casi).** *Rev. Internat. du Trach.*, Avril, 1933.

(3) **Busacca** has written an interesting clinical study of a hundred cases of trachoma which should be read in the original by those who live in countries where this disease is rife. His most important point is that trachomatous pannus constantly can be detected by the use of the slit-lamp in the earliest cases. In his opinion the corneal rosettes and peripheral pits of Herbert are found only in the lunular part of the conjunctiva which covers the cornea and not in the real corneal tissue. The depressed scars which are to be found in many cases anywhere on the cornea, which are the result of infiltrations, have a different origin from the peripheral pits; an opinion which would not be universally accepted.

A. F. MACCALLAN.

(4) **Cattaneo (Sassari).—Unilateral trachoma. (Sul trachoma unilaterale).** *Rev. Internat. du Trach.*, January, 1932.

(4) Among 1,600 trachoma cases treated by **Cattaneo** he has found 16 in which only one eye appeared to be affected. However, on examination with the slit-lamp all were found to have both

eyes affected. For many years the reviewer has urged the importance of such an examination. This does not mean that unilateral trachoma is a non-existent phenomenon but that adequate examiation reduces the number of these cases.

A. F. MACCALLAN.

(5) **Wilson, Rowland P. (Cairo).—A short slit-lamp study on the corneal vessels in Egyptian trachoma with a discussion on their diagnostic value in doubtful cases.** *Folia Ophthal. Oriental.*, Vol. I, Fasc. 1, p. 52.

(5) **Wilson** points out the scant attention that has been paid to the formation of vascular loops and capillary end loops at the limbus in the normal eye and particularly so in the study of trachoma with vascularization of the cornea and pannus.

He describes three vascular zones at the limbus: the palisade zone, zone of vascular loops, and the zone of end-capillary loops; also a primary groove in the palisade zone and at the junction of sclera and cornea and a secondary groove at the junction of the vascular loops and end-capillary loops. In trachoma the vascular changes take place in the vicinity of the secondary groove, the end-capillary loops expanding and extending into the cornea instead of occupying a narrow fringe and being disposed in a close network as under normal conditions.

Diagrams and coloured slit-lamp drawings illustrate these changes. Wilson comments on the early stage at which corneal vascular changes are evident. Many cases of trachoma in Egypt occur during the first year of life and typical vascular changes have been seen in the cornea one month after infection. Points in differential diagnosis are discussed.

H. B. STALLARD.

(6) **von Taborisky, J. (Haifa, Palestine).—Ueber die mikroskopische Diagnose des Trachoms.** *Folia Ophthal. Oriental*, Vol. I, Fasc. 1, p. 34.

(6) **von Taborisky** states that bacteriological, cytological, and histological examinations are important in the differential diagnosis of trachoma. The micro-organisms found in the conjunctival sac in cases of trachoma do not differ from those present under normal conditions.

Scrapings of the conjunctiva show alterations in the epithelial cells; the cylindrical cells of the palpebral conjunctiva are transformed into squamous cells and the nuclear membrane of these cells shows a jagged outline and later the nucleus undergoes complete degeneration. Inclusion bodies in the conjunctival epithelium can be found early in the disease, but after it has been established for one year, these are often untraceable.

Thus in the early stages of trachoma the examination of epithelial scrapings is of value, for in no other inflammatory disease or degeneration of the conjunctiva are inclusion bodies to be found. Transformation, proliferation and degeneration of the epithelium are characteristic of trachoma. The structure of the follicles differs in site and histological appearance from that of chronic follicular conjunctivitis.

H. B. STALLARD.

(7) **Sobhy, Mohammed Bey** (Cairo).—**The true nature of post-trachomatous degenerations** (**P.T.D.**). *Bull. Ophthal. Soc. of Egypt*, 1932.

(7) "In all forms of inflammatory conditions of the conjunctiva of some standing, the epithelium is thrown into papillae, the size of these differ in every individual case. Even in the initial stages of trachoma (MacCallan, Tr. I.) these papillae are evident microscopically, although they are not seen clinically (naked eye appearance). As a result of this papillary hypertrophy the spaces between the papillae are lined with epithelium, and look in section like glandular tubules. By further evolution of the trachomatous process the subepithelial layer increases in thickness and the pseudo-glandular tubules send in further deeper epithelial plugs, similar to glandular cancer. The epithelium of the blind end of these plugs cannot be shed off on the surface, and will accumulate inside the tubule as epithelial débris mixed with leucocytes, which cannot be discharged through the small openings or mouths of the pseudo-glandular tubules or epithelial down-growths; as a result of this the tubules are transformed into cysts filled with inspissated material that may undergo calcareous degeneration. This is the true nature of the so-called post-trachomatous degenerations as described in all the books of pathology of the eye, and called by some concretions or concrément. Dr. MacCallan in his early days in the Egyptian Ophthalmic Hospitals, as a good clinical observer and in the absence of histological preparations, described them as degenerations of the trachomatous follicles. In fact they are so intimately connected with trachoma and I advise him to give them the name of 'Post-trachomatous concretions' (P.T.C.), so as not to bring much change in the first name put down by one of the masters of ophthalmology in Egypt, and in commemoration of his stay among us."

During the discussion on the paper Wilson expressed his agreement with **Sobhy** and stated that he had previously published histological evidence on this subject.

The reviewer accepts with pleasure the pathological correction and the kind compliment, but he sees no reason why what Sobhy

himself calls a calcareous degeneration should be renamed a concretion. The term "Post-trachomatous degeneration" or "P.T.D." has become known to hundreds of ophthalmologists and should not be disturbed.

A. F. MACCALLAN.

III.—LENS

(1) **Gourfein, D. (Geneva).—Senile cataract and the metabolism of calcium. (Cataracte senile et metabolisme du calcium).** *Rev. gen. d'Ophtal.*, February, 1932.

(1) **Gourfein**, after a review of various theories as to the aetiology of senile cataract, discusses the influence of calcium deficiency. Through his *chef de laboratoire*, Piotrowski, he made experiments to compare the total blood calcium in cataractous and non-cataractous individuals and gives detailed tables showing the results. Since "lesions of the crystalline lens" in tetany have been attributed to deficiency of blood calcium he takes notice of this also. Gourfein's conclusions are: (1) There is no difference between the total calcium content of the serum in 21 cases of cataract and 26 non-cataractous cases. (2) In no case (except one non-cataractous) are there spontaneous signs of tetany (as investigated by the signs of Chwostek, of Trousseau, of Lust, and of Weil). (3) There is no relation between the proportion (*taux*) of calcium and stage (*degré*) of the cataract.

ERNEST THOMSON.

(2) **Riddell, W. J. B. (Glasgow).—On the complaints of patients with senile cataract.** *Glas. Med. Jl.*, May, 1933.

(2) The short article by **Riddell** is excellent, within the scope of the author's intentions. It presents to the general practitioner the various complaints made by patients with various types of cataract and relates these complaints in a general way to the kind of cataract. It cautions the practitioner on the various ways in which cataract may be secondary to other diseases, or may exist in association with them, so altering the prognosis. The very difficult question of what and how much to say to the patient is fairly fully discussed and particular attention is paid to the necessity for caution in using the words "cataract" and "operation," and it is pointed out that, in the minds of some patients, cataract and cancer are synonymous terms. As doubtless, the author would be the first to admit, a full synopsis of his very helpful article would be out of place in a specialist journal.

ERNEST THOMSON.

(3) **Whiting, Maurice M. (London).—Modern development in cataract extraction.** The Montgomery Memorial Lecture, delivered at the Royal College of Surgeons in Ireland, February 17, 1933.

(3) **Whiting,** in the Montgomery Memorial Lecture, described the methods of intra-capsular cataract extraction as practised by Smith, Barraquer, and those who use the capsule forceps for delivering the lens in its capsule. It is evident that Whiting favours the extra-capsular method of extraction with iridectomy and subsequent capsulotomy when indicated. The necessity of a bacteriological culture before operation is stressed, also the need for careful medical treatment of cases of diabetic cataract. He described the technique which he believes to be safe for the patient and discusses the advantages and disadvantages of certain technical modifications such as seventh nerve block, retro-ocular anaesthesia, a suture passed through the superior rectus, capsulotomy simultaneously with the corneal section, cystitome or capsule forceps, and other matters. The lecture contains much sound practical advice about cataract extraction from a surgeon of wide experience and conservative views.

H. B. STALLARD.

(4) **Saradindu Sanyal (Calcutta).—A preliminary note on the clinical use of insulin in the anterior chamber haemorrhage after cataract extraction.** *Calcutta Medical Journal*, March, 1933.

(4) **Saradindu Sanyal** reviews the causes and varieties of intra-ocular haemorrhage during and after cataract operations. For the small amount of blood that enters the eye from the corneal section and margins of the iridectomy he recommends the internal administration of calcium glycerophosphate 15 grs. with parathyroid 1/10 gr. twice a day. For expulsive haemorrhage occurring immediately after the corneal section is made, the author advises that the operation be abandoned, the eye firmly bandaged, ice applied to the neck and atropine and morphine injected subcutaneously. In arterio-sclerosis it may be necessary to perform venesection. Takahasi has suggested that in diabetes, insulin by lowering the blood sugar, increases the fibrin ferment present in the aqueous. Saradindu Sanyal has attempted to put this to practical use in cases of haemorrhage into the anterior chamber. Insulin is given in small doses, commencing with two units and gradually increasing the dose. The injection is made before a meal. A rich carbohydrate diet is given and the blood sugar estimated at three hourly intervals after the meal. Precautions are taken against hypoglycaemia and special care is necessary in cases of elderly persons, arterio-sclerosis, cardiac disease, and

gastro-intestinal disorders. The author claims that the administration of insulin accelerates the absorption of blood clot in the anterior chamber. A complication occurred in one elderly patient submitted to this treatment. After five injections of two units of insulin the vision in his better eye was reduced from 6/6 to 6/24, but subsequently recovered seven days after the insulin injections were omitted. The urine was sugar free. The author believes that this was due to changes in the water balance of the body induced by insulin. The author states that the number of cases recorded is at present too small for any definite conclusions to be made, but, nevertheless, it is a fact that insulin accelerates the absorption of blood in the anterior chamber.

H. B. STALLARD.

(5) **Green, John and Beisbarth, Carl (St. Louis).—Extraction of congenital and young adult cataract by the method of Barkan.** *Amer. Jl. of Ophthal.*, July, 1933.

(5) Green and Beisbarth review briefly the disadvantages and complications arising from the needling operation for congenital and juvenile cataracts and of cataract in early adult life, whether traumatic or not. They describe the technique elaborated by Otto Barkan for "the extraction of congenital soft and membranous cataracts." Full dilatation of the pupil is essential and this is obtained by the subconjunctival injection of adrenalin 1/1,000. By this means the pupil remains fully dilated when the anterior chamber is lost. An incision is made in the cornea with a keratome 2 mm. from the limbus, the keratome being entered very obliquely so as to produce a valve-like opening which is quickly sealed. Through this opening a cystitome, capsule forceps, hook, spoon, irrigator, and even scissors may be passed as required. The authors state that the amount of manipulation possible is surprising and there is no tendency of the well-retracted iris to prolapse. Six case reports are given.

H. B. STALLARD.

IV.—MISCELLANEOUS

(1) **Conway, J. A. (Glasgow).—Case of double endophthalmitis complicating pneumonia.** *Glasgow Medical Journal*, October, 1932.

(1) Conway's case is that of a miner who was admitted to hospital with double endophthalmitis. Fourteen days previously he had developed left lobar pneumonia and twenty-one days

previously his left little finger had been severely crushed. On admission the left eye was in a more advanced state of purulent disorganization than the right. Orbital cellulitis was present, yet investigations failed to show any cause other than the pneumonia. Pus withdrawn from one of the eyes showed numerous polymorphonuclear pus cells and Gram-positive diplococci, a few of which were intra-cellular. Both corneae perforated and the eyes shrivelled. Eight weeks after admission the patient was allowed to go home, there being no contraindications. "Two days later he complained of a sudden pain over the heart, and died shortly afterwards." The author does not mention an autopsy and the presumption is that none was held, but he adds the following significant sentence: "It would seem that the endocardium may become involved at a comparatively late date, and the lesson I have learned is that these cases require a long convalescence in bed as a precautionary measure. The case described showed no signs of acute pyaemia or septicaemia: nevertheless, a fatal complication developed."

ERNEST THOMSON.

(2) **Giannantoni (Perugia).—The nature of amblyopia from lead-poisoning. (Ricerche sperimentali sull'ambliopia tossica saturnina).** *Boll. d'Ocul.*, February, 1933.

(2) Lead is one of the commoner causes of toxic amblyopia. Its effect is much more serious than that due to tobacco, and the pathology is not well understood. With the view of throwing light on the mechanism **Giannantoni** has made a series of experiments on rabbits; these were given doses of lead, in some cases by the mouth, in others by subcutaneous injections. The animals sometimes survived for many months.

As a result of the examination of the retinae and optic nerves the author concludes that both the retina and the nerve are affected by the poison. The retina shows marked degeneration of the ganglion cells; the nerve shows first breaking up of the myelin sheath and then degeneration of the axis cylinder; the changes in the glia and in the vessels are often wanting and when present only slight.

HAROLD GRIMSDALE.

(3) **Benjamin, C. E. (Vienna).—A cure of cavernous sinus thrombosis. (Una curación de trombosis en el seno cavernoso).** *Rev. Oto-Neuro-Oftal. y de Cir. Neurol.*, Vol. VII, p. 127, April, 1932.

(3) After an anatomical consideration of the connections of the cavernous sinus **Benjamin** gives a short résumé of the symptomatology and causation of its thrombosis. The prognosis

being uniformly bad, heroic surgical measures are sometimes resorted to. Brunner reported cure of a unilateral case after ligature of the internal carotid, Bircher after opening the sinus through the orbit, while fairly recently Christopher has had a successful case in which he drained the sinus by means of Krönlein's operation. Benjamin's case was a boy, aged 15 years, suffering from a right-sided purulent otitis media. A right-sided mastoid operation was performed. The patient developed many secondary abscesses. Twelve days after operation inequality of the pupils was noticed—four days later there was proptosis. Abscesses formed in both orbits, which were opened. The process subsided, but further secondary abscesses occurred: parotid, pharyngeal, etc. The patient finally recovered with a unilateral optic atrophy.

IDA MANN.

BOOK NOTICES

Palestine Department of Health, Annual Report, 1932. Ophthalmic Service. Printing Office, Jerusalem.

There are now seven special ophthalmic clinics maintained by the Department in Beersheba, Jaffa, Gaza, Ramleh, Nablus, Tulkarem and Acre. The most serious ophthalmic question which has to be dealt with in Palestine is the summer epidemic of acute conjunctivitis which is rife in the southern districts. According to the 1931 Census 39 per cent. of the population in this part of the country were blind in one or both eyes. This almost incredible ocular devastation has been corroborated by Dr. Strathearn who spent a month in the villages most affected. It is proposed to re-establish a mobile travelling ophthalmic unit to visit the scenes of the more severe epidemics.

However the main causes of the occurrence of these epidemics and of their severity are a greatly diminished average rainfall for several years and an increasing population. With a grossly insufficient water supply, providing not even a minimum for domestic purposes, ocular conditions, in the opinion of the reviewer, are likely to get worse even though increased opportunity of attending an ophthalmic clinic is provided. While the drastic methods of Herod are unlikely to appeal to the very able Director of the Department of Health, Colonel Heron, some diminution in the increase of the population could be affected by stopping all immigration into Palestine until the rainfall again became normal, for in the six months, January to June, 1932, 4340 persons came to reside in the country.

CORRESPONDENCE

A NEW FORCEPS FOR INTRACAPSULAR EXTRACTION OF CATARACT

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—It was rather amusing to me that while I was writing down some notes regarding the new use of an old forceps, in the operation of intra-capsular extraction of cataract, I read in the last number of the Journal (August, 1933) almost similar notes on the subject. I have not yet extracted as many cataracts with this forceps as has Mr. R. B. Ratnakar, and I was postponing the publication of these lines till I had operated on more cases.

The use, for the first time, of this forceps which I endeavour to describe now, occurred to me accidently, but further practice has approved its subsequent use.

Our historic Qalawoon Hospital (founded in 1284 A.D., long before the elaborate system of modern hospitals of the era of MacCallan), for a time previously staffed by distinguished oculists belonging to a French School, has amongst its instruments some very good French ones. The forceps above mentioned, belongs to this category of instruments. It is illustrated in Luer's catalogue under the name of *Pince à cuillers du Dr. Schmidt*, and in the American Encyclopedia of Ophthalmology it is described (page 5263) as "Schmidt's Spoon Forceps, for the extraction of Lenticular Remains." In our hospital it is called the "Clot Forceps," and I always have it at hand for every intra-ocular operation.

In 1931, I brought with me from Vienna a Kalt forceps for extracting cataract in its capsule. I left it in the hospital for occasional use. Before leaving for the Congress in Madrid, 1933, I collected and took back to my private clinic the few instruments which I kept in hospital for my personal use. On resuming work I forgot to take with me to hospital the Kalt forceps. This I discovered while operating on a cataract and after the section was made. I attempt the intra-capsular method in every suitable case, and owing to the absence of the forceps reserved for this purpose, I ventured to try this so-called "clot forceps," and to my amazement the lens came out very easily and the operation

ended uneventfully. I am now using it instead of the Kalt forceps. The points to note about this forceps are the following:—

1. The extremities of the forceps end in spoon-like concavities which come in apposition all round the edge when the forceps is closed. In this way it differs from Mr. Ratnakar's forceps, in that the knuckle of capsule caught up by the forceps cannot be seen through the cornea being enclosed in the concavities.

2. The advantages of a forceps ending in such a hollowed fashion, either like Mr. Ratnakar's or the one I describe, is that the capsule tissue pressed between the two ends of the forceps, the greater part of which finds shelter or protection in the mentioned concavities, is less likely to be crushed or torn out, as it would be with a forceps whose ends meet in every point as is the case in Kalt's forceps.

3. The application of other capsule forceps curved to fit the anterior surface of the lens should be very dexterously done, more so than with this forceps which meets the lens in a smaller extent and so can grasp the lens where it meets it.

Yours truly,

M. KHALIL.

15A, MADABEGH STREET,
CAIRO.

NOTES

Appointment

Mr. ARNOLD SORSBY has been elected Hunterian Professor at the Royal College of Surgeons for 1933-34. He will deliver his lecture, the subject of which is Retinal Abiotrophy, on Monday, January 29, 1934, at 5 p.m.

* * * *

The William Mackenzie Medal

THE William Mackenzie Medal will this year be awarded to Professor Jules Gonin of Lausanne in recognition of his valuable contributions to ophthalmology, especially those bearing on the treatment of detachment of the retina. The presentation of the medal will be made in the hall of the Royal Faculty of Physicians and Surgeons, Glasgow, on Friday, October 13, at 8.30 p.m., when Professor Gonin will deliver the William Mackenzie Memorial Lecture on "The Evolution of Ideas concerning Retinal Detachment within the last five years."

National Society for the Prevention of Blindness, Inc. U.S.A.

IN May, 1933, the National Society for the Prevention of Blindness celebrated its 25th anniversary. The report of the proceedings gives an account of the increase in membership of the society from 10 to 16,600; and of its collaboration with the medical profession in preventive work, the education of visually handicapped children and eye accidents occurring in industry. Special mention is made of preventive work in ophthalmia neonatorum, dangerous toys used in child play and industrial accidents. There is an account of the increase in number of sight-saving classes from two in 1913 to 421 in 1932. Mention is made of special clinics for strabismus cases in New York, Philadelphia, and Cincinnati.

* * * *

The Sight-Saving Review

PUBLISHED by the National Society for the Prevention of Blindness. Inc. U.S.A., Vol. III, No. 1, March, 1933.

"Sight-saving" work in 1932 is reviewed particularly in regard to prophylactic work in eye accidents during child play and in certain industries; in ophthalmia neonatorum and syphilis. The necessity of pre-school vision testing, clinics for strabismus cases and ophthalmic hygiene in schools is discussed.

A paper relates the diagnostic importance of an ophthalmological examination in the discovery of certain constitutional disorders. The history and development of sight-saving classes in the United States is described and an outline is given of the scope of social service among eye patients.

Vol. III, No. 2, June, 1933.

In an article on "Diet and Eye Health," the author points out that "many eye troubles are due to upsets in the physico-chemical equilibrium," and stresses the importance of minerals, such as calcium and of vitamins in the diet.

In another article it is urged that applicants for motor drivers' licences should receive a more thorough test of vision than exists at present. A paper is devoted to trachoma in the native white population of the United States. The authors describe the routine work in their trachoma hospitals, the differential diagnosis of this disease and the indications for various surgical operations. Another paper is concerned with the reading ability of sight-saving class pupils in Cleveland, Ohio.

* * * *

Course in Ophthalmology and Otolaryngology, 1934

THE Research Study Club of Los Angeles will present its third annual mid-winter course in ophthalmology and otolaryngology January 15 to January 26, 1934, inclusive. The guest lecturers in ophthalmology will be Professor A. Elschnig, of Prague, and Dr. Hans

Barkan, clinical professor of ophthalmology of Stanford Medical School, San Francisco, California. In otolaryngology the guest lecturers will include Dr. John Barnhill of Miami, Florida; Dr. E. C. Sewall, of San Francisco, California; and Dr. Harry L. Baum, of Denver, Colorado. The fee for the entire course will be fifty dollars. For further information and a detailed programme address the secretary, Dr. Don Dryer, 2007 Wilshire Boulevard, Los Angeles, California.

FUTURE ARRANGEMENTS

1933

October 6.—Midland Ophthalmological Society, Annual Meeting, at Birmingham and Midland Eye Hospital.

October 20.—North of England Ophthalmological Society, at Manchester.

November 17.—North of England Ophthalmological Society, at Bradford.

December 5.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital.

December 15.—North of England Ophthalmological Society, at Leeds.

1934

February 16.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

March 16.—North of England Ophthalmological Society, at Liverpool.

April 13.—North of England Ophthalmological Society, at Sheffield.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *August, 1933.*

TRONCOSO. The continuous circulation of the aqueous humour.

ABRAMSON and TEITELBAUM. The Argyll Robertson phenomenon in multiple sclerosis.

CHARLIN. Simulation and ocular hysteria.

FRY and BARTLEY. The brilliance of an object seen binocularly.

LEVITT. Recurrent vitreous haemorrhage.

CLARK. Entoptic phenomena observed in my eyes before and after cataract operations.

DE LONG. Metastatic carcinoma of the choroid.

HOFFMAN. Medical economics.

REESE and GUY. Exophthalmos in leukaemia.

FEJER. Cholesterin crystals in the anterior chamber.

Archives of Ophthalmology. *August, 1933.*

NAKAMURA. Specific treatment of ophthalmic tuberculosis with a new tuberculosis vaccine.
FERREE, RAND and SLOAN. Selected cases showing the advantages of a combined tangent screen and perimeter.
SAMUELS. Sympathetic scleritis.
HUGHES. Removal of the lid with plastic repair.
HARE and DYKE Röentgen therapy of pituitary tumours: report of twenty cases.
VERHOEFF. A new test for visual acuity.
BURNHAM. Respective values of the various forms of treatment of certain diseases of the cornea.
HOBART. Protein extract of vitreous humour. Preliminary report.

Annales d'Oculistique. *August, 1933.*

MICHAIL. Cystic angioma and lymphangiectasis of the orbit.
ANNEN. Sarcoma of the choroid.
ROBERT. The influence of ametropia on certain infections of appendages of the eye (lids, lacrymal passages).
GABRIÉLIDÈS. About a micro-organism met with for the first time on the human conjunctiva.

Archives d'Ophtalmologie. *August, 1933.*

VAN DER STRAETEN and MALDAGUE. Four cases of disease of the suprarenal capsule with ocular symptoms.
VILLARD and DEJEAN. Morphological study of pterygium by the slit-lamp. Pathogenic deductions.
BEAUVIEUX. Treatment of follicular tuberculosis by methylique antigen.
FRANÇOIS. Chronic oedema of the cornea.

Revue d'Oto-Neuro-Ophtalmologie. *July-August, 1933.*

COPPEZ. Some rare cases of ocular synkinesis.
SÉDAN. Note on the chronological order of nerve compressions in tumours of the orbital apex.

Klinische Monatsblätter für Augenheilkunde. *August, 1933.*

VON BLASKOVICS. Improvement of the suction operation for cataract.
KUNZ. The occurrence of iritis with chronic inflammation of the vertebral column.
RIECHERT. Retinitis pigmentosa and multiple sclerosis.
KOYANAGI. Morphological blood changes in renal retinitis.
BRAUN. Contribution to the question of maculo-cerebral types of hereditary degeneration of the macula lutea.
VOS. Myotonic dystrophy with cataract.
KREIKER. A rare abnormal development: cilia under the conjunctival fold.
RÖTTH. The significance of displacement of the lid margins in the aetiology of conjunctival and corneal inflammation.
SPINELLI. Recanalization of the lacrymal ducts by diathermy.
ASCHER. The connection between intra-ocular pressure and bulbar volume.
JANSON. Observations on ocular leprosy in Lettland.
WOLFSOHN-JAFFÉ. Hereditary anterior capsule cataract.
GOLDMANN. Some notes on Vogt's "new kind" of cataract in albinos and on his fundamental experiment in the biology of ultra-red cataract.
SZILY. Comparative examination of the efficacy of the various methods of treatment of retinal detachment by animal experiment.
HARTMANN. Peritomy for the prevention of gonococcal corneal complications.
AMMANN. Traumatic keratitis superficialis-interstitialis.

Annali di Ottalmologia e Clinica Oculistica. *April, 1933.*

GIANNINI. Clinical contribution to the study of familial amaurotic idiocy and the analogous hereditary degenerative form.
RINALDI. Clinical research on the property of antigen in the uveal pigment.
BIFFIS. Report on calcium and potassium in the blood of the glaucomatous.
RINALDI and QUAGLIO. Intra-dermal reactions with uveal protein in affections of the uvea.

Archivio di Ottalmologia. *April, 1933.*

PADOVANI. A case of high hypermetropia.
VITTADINI. Contribution to the knowledge of the histo-pathological changes of the lacrymal sac in chronic dacryocystitis.
BARATTA. Comparative histological research on the principal methods of tattooing for corneal leucoma.

Bollettino d'Oculistica. *July, 1933.*

STRAMPELLI. Treatment of retinal detachment with sub-retinal injection of blood plasma.
WAARDENBURG. On the anomalous length of the lacrymal canals associated with lateral displacement of the medial canthus and hyperplasia of the inter-ocular regions and other defects.
KOCH. On the destruction of the lens fluorescence.
VELE. On congenital coloboma of the iris and choroid.
VITTADINI. A fatal case of staphylococcal septicaemia following hordeolum internum.
DE CRISTOFARO. A particular case of transparent marking of the upper lid.
VENCO. Ocular complications in a case of spirochaetal haemorrhagic jaundice.

Revista Oto-Neuro-Ottalmologica e Radio-Neuro Chirurgica. *May-June, 1933.*

CARDILLO. Clinical and radiological review of a case of Erdheim's tumour.
BUSACCA. Three cases of papilloedema of difficult interpretation.
——— Tumour of Rathke's pouch.
SPINELLI. Retinal arterial pressure in relation to intra-cranial hypertension and variations following endo-cranial interventions.

Archivos de Oftalmología Hispano-Americanos. *August, 1933.*

COURTIS. Some anatomo-pathological considerations on arterio-sclerotic retinitis.
CARRERAS. A case of Mikulicz's syndrome.
SCHOUTE. The artificial eye in Spanish literature.

Revista Oto-Neuro-Oftalmologica y de Cirujia Neurologica Sud-Americana. *August, 1933.*

PAVIA. Photograph of the luminous slit-lamp beam in the human eye.